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SOME COMMON BACTERIA AS SHOWN BY THE ELECTRON MICROSCOPE

A Preliminary Report

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An electron microscope has been installed in the Institute of Nuclear Physics, Calcutta University. The technical features of this new microscope have been published elsewhere. (Das Gupta *et al*, 1948). An account is given here of the electron microscopic study of some common bacteria, *viz.*, Bact. coli, Streptococus hæmolyticus and Leptospira icterohæmorrhagia which have been photographed with this instrument. All these photographs were taken with 50 kv. electrons without any limiting aperture in the objective lens.

Operating Principle of Electron Microscope

The electrons emitted from a hot tungsten filament are accelerated by a potential difference of 50,000 volts. The electron beam then passes through a magnetic field which acts as a condenser lens and causes the electrons to follow nearly parallel paths along the axis of the instrument. A very narrow electron beam then passes through the specimen supported very close to the objective pole pieces and thence through the electromagnetic objective and projective lenses and finally forms a magnified image of the specimen on a fluorescent viewing screen or photographic plate.

Any electron that is scattered or deflected out of its path so that it fails to enter the small objective aperture and is unable to reach the final screen or photographic plate, cannot contribute to the brightness of the image; while those electrons which pass undeflected contribute to the general brightness. The final image is therefore a record of the amount of electron scattering at each point of the object. Points of the object appear dark in proportion to the number of electrons they scatter and the final image appears dark against a bright background formed by unscattered electrons.

As air itself would scatter most of the electrons out of their path, it is necessary to create an anærobic condition in the whole microscope as far as practicable. For this purpose the gas pressure inside the microscope is reduced to about 0·1 microns by means of a pumping system consisting of an electrically heated oil diffusion pump backed by a high speed rotary oil pump. The bacterial as well as other specimens are introduced into the microscope mounted on a very thin collodion film (thickness of the order of 10 millimicrons) stretched over a fine wire mesh in a holder. These supporting films are so thin as not to obstruct the electrons and are sufficiently strong mechanically at the same time to withstand exposure to intense electron bombardment for a considerable time. Specimens thicker than about one micron cannot be studied satisfactorily in this manner except when higher accelerating voltages are used as then the excessive volume scattering of electrons within the thick object cause the image to become diffuse and hazy.

The smallest detail that may be revealed by any microscope is known as its limiting resolution and is given by the formula $\sqrt{d_{min}} = \frac{\lambda}{2n \sin \alpha}$

where λ is the wavelength of the illuminating radiation, n the refractive index and 2α is the divergence of the allowed pencil of rays. With oil immersion objectives, $n = 1 \cdot 5$, α is close to 90, so that $d_{min} \simeq \lambda/3$. For visual examination and the smallest theoretically distinguishable detail is of the order of 170 millimicrons. Assuming resolving power of the unaided human eye to be approximately 0·2 mm or 200 microns, a magnification of 1200 X is sufficient to make these details visible.

The electron radiation when accelerated by 50,000 volts has a wavelength 100,000 times smaller than visible light suggesting the possibility of much improved resolution in a microscope using electron beam in the place of visible light.

Experimental

The plates reproduced herein show the electron micrographs of some common bacteria. Fig. 1 shows

Streptococcus haemolyticus grown 36 hours on blood agar. A plate culture was made from which suitable colonies were selected and subculture made on slanting blood agar medium. The cell walls of the oval cocci can be seen to extend to form a short chain. Longer chains of variable lengths as well as discrete groups were observed visually. The final magnification is 29,500 X.

Fig. 2 shows Bact. coli grown 19 hours on agar slants. A plate culture was made, suitable colonies were selected and subculture was made on agar slant medium. It is to be noted that all the bacteria photographed were dead due to their being in the high vacuum and being exposed to electron bombardment inside the microscope. A certain sample also showed the presence of cocco-bacillary forms. No flagella are however evident in the picture. The magnification in this case is 13,000 X.

The sample of Leptospira icterohaemorrhagiae, shown in Fig. 3 was grown for 7 days in Vervoot's medium containing blood serum. The characteristic hooked ends of the leptospira are evident in the picture, and the spirals which are normally seen with ordinary optical microscope under darkground illumination from fresh specimens are also evident to some extent. This decrease in undulations is probably due to the straightening out of the organism by dehydration which occurred during prolonged exposure to high vacuum inside the microscope. The scattered dark specks all over the field are due to protein impurities and other foreign materials in the culture medium. The magnification in this case is 15,200 X. In all micrographs, magnification is indicated by a straight line representing one micron in length.

Coclusion

In conclusion it is to be noted that the above micrographs were taken at the preliminary stages of adjustment of the new instrument. They however amply demonstrate the usefulness of the electron microscope in investigating detailed morphology of bacterial specimens.

Acknowledgment

The authors wish to thank Prof. M. N. Saha, F.R.S., for his great interest in this work and Dr. N. N. Das Gupta for constant help and encouragement. They are also indebted to the Director, School of Tropical Medicine, Calcutta for providing bacterial cultures and to the Council of Scientific and Industrial Research for financial assistance.

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INOSITOL IN PHOSPHOLIPIDS

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Phospholipids represent a family related to fats. Besides fatty acids and glycerol, they contain some of the other substances like phosphoric acid, choline, *inositol*, galactose, ethanolamine and tartaric acid, attached to the glycerol part of the molecule. They are found in all animal and vegetable cells. Brain, heart, kidney, liver, spinal cord, eggs, tubercle bacillus, and soyabeans are rich in phospholipids. They play an important rôle in the metabolism of fats. Inositol is hexahydroxycyclohexane.

Nine different cis-trans isomers of inositol are possible [1](Rosenberg, 1945). Not all forms of inositol are biologically active. Meso-inositol or *i*-inositol which is optically inactive, is biologically active. Naturally it has been found to occur in four different ways[2], which are as follows:

1. Free inositol
2. Phytin
3. Lipositol
4. A non-dialyzable and water-soluble complex.

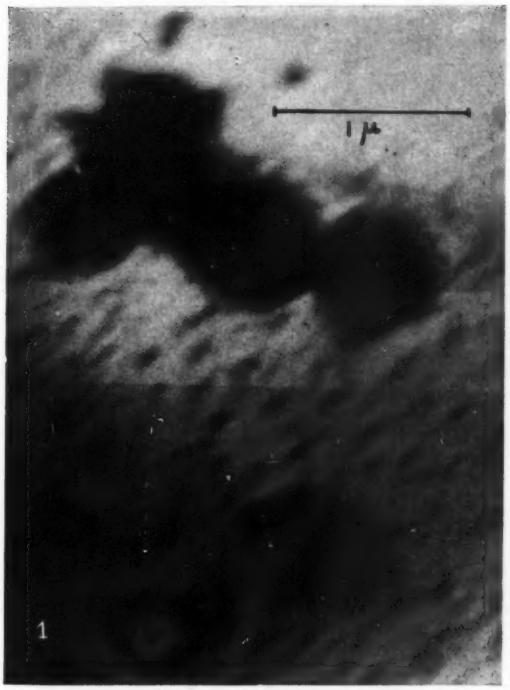
Free inositol occurs in all the living cells.

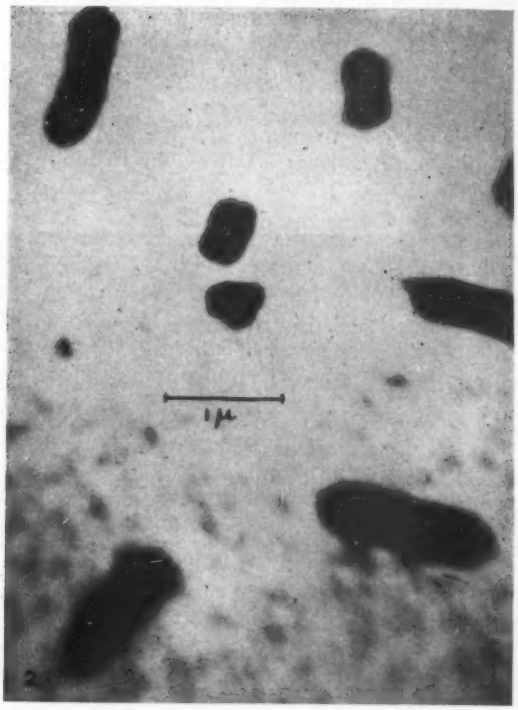
Inositol in Phytin

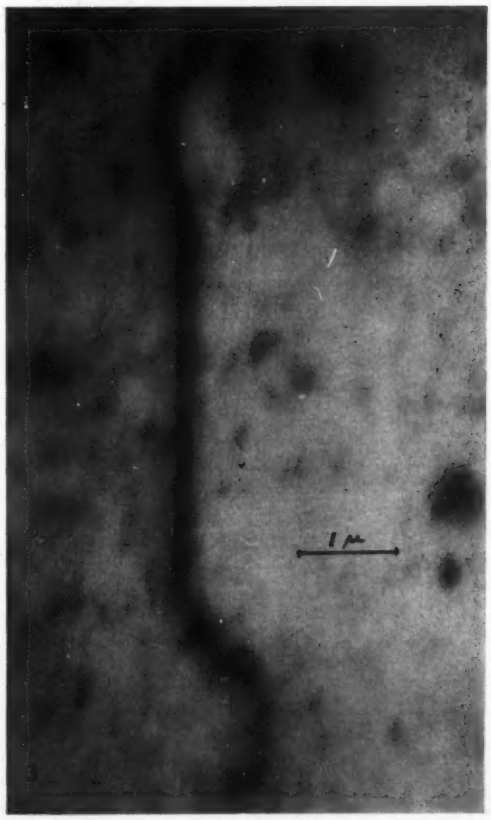
Phytin is the calcium magnesium salt of inositol-hexaphosphate. It occurs in many seeds and its occurrence in plant kingdom is known for a long time. Rapoport[3] (1940) was the first to identify the occurrence of phytic acid in animal kingdom. He isolated is from erythrocytes cells of the blood of chickens, other birds and turtle. He isolated inositol from barium phylate by hydrolysis with (2 N) H_2SO_4.

Inositol in the Phospholipids of Human Tubercle Bacilli

Anderson[4] (1930) noted the occurrence of inositol in the phosphatides from human tubercle bacilli. Acid (dil. H_2SO_4) hydrolysis of the phosphatide yielded 67 per cent fatty acids and 33 per cent water-soluble material. The water-soluble material was found to contain glycerophosphoric acid, mannose phenylhydrazone, glucosazone and small quantity of crystals of inositol. From the phosphatides inositol was separated according to the following scheme:







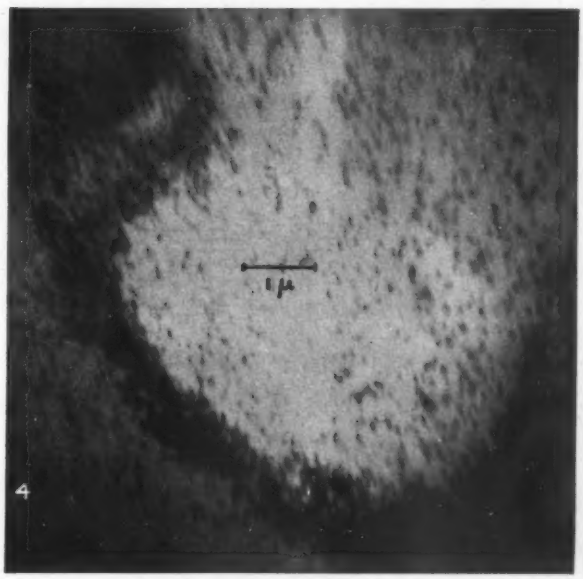
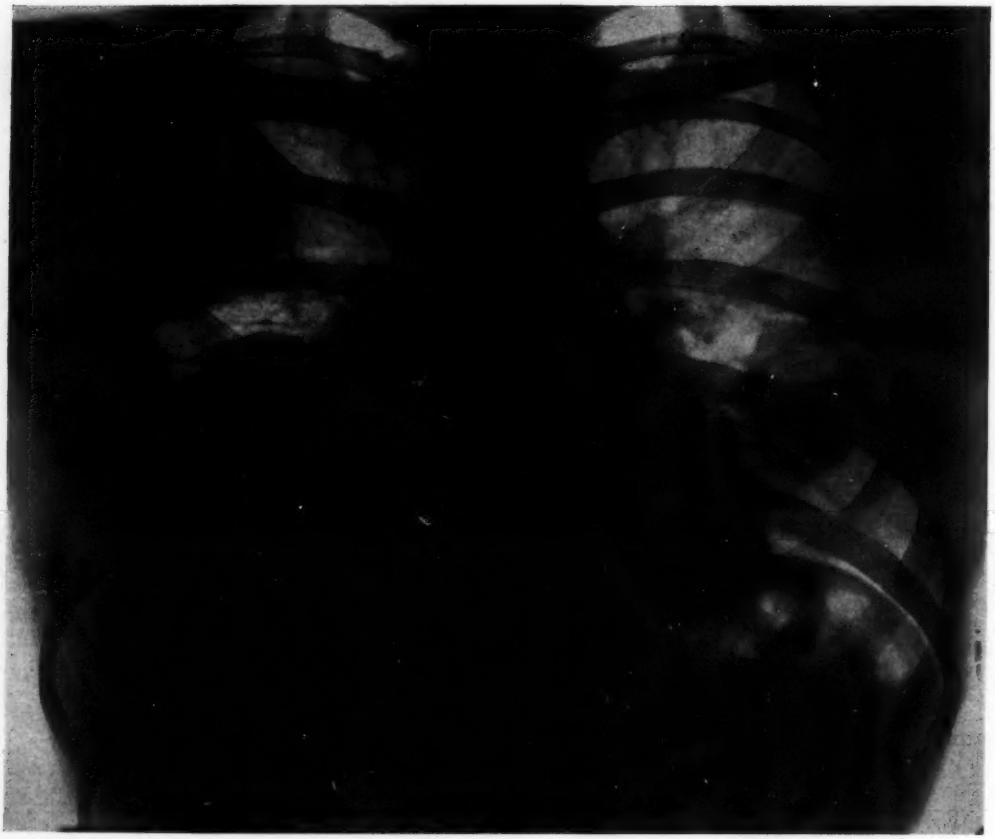
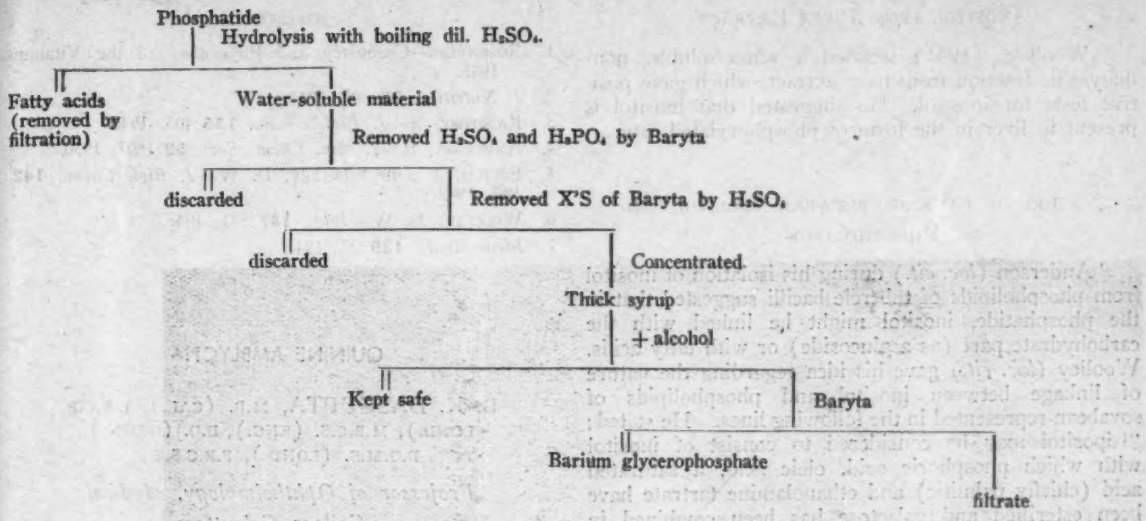


FIG. 1—Streptococcus Hæmolyticus.
FIG. 2—Bact. Coli.
FIG. 3—Leptospira Icterohæmorrhagiæ.
FIG. 4—Leptospira Icterohæmorrhagiæ.



Skiagram showing Two Cavities with Fluid Level and an Irregular Dense Opacity at the Right Base



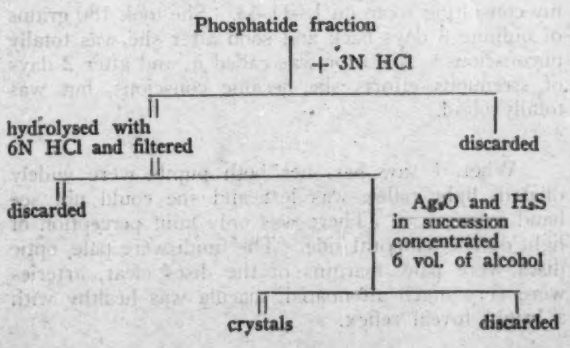
From the filtrate excess of barium was removed by sulphuric acid and the solution was concentrated. To the concentrated solution was added the portion insoluble in alcohol (kept safe). The whole thing was then concentrated in vacuum. Crystals of inositol (or inosite) were obtained, as identified by a positive Scherer test and M.P. 224-25°c.

Inositol in the Phospholipids of Nerve Tissue

Folich and Woolley[5] (1942) isolated inositol from the phosphatides of brain and spinal cord. They pointed out that brain cephalin is rich in inositol. They obtained the phosphatide fraction by repeated precipitation of the chloroform solution with alcohol. It was then dialyzed and the fraction least soluble in alcohol contained inositol. It was 0·4 per cent of the net weight of the brain and contained 6·8 per cent inositol.

Separation of Inositol

From the phosphatide fraction, they isolated inositol by the following scheme:



The crystals were recrystallized from alcoholic HCl and were identified as inositol, M.P. 217°, Hexa-acetate M.P. 211-3°C.

Inositol in Soyabean Phospholipids

Woolley[6] (1943) isolated inositol from soyabean phospholipids, which he called *Lipositol*. He found that this new phospholipid contained the following constituents shown in the Table I.

Constituent	*Found by isolation*	*by analysis*
Inositol ..	.. 15%	16%
Galactose ..	.. ..	05·5%
Tartaric acid	.. 8·3%	..
Ethanolamine	.. 0·44%	..
Oleic acid ..	.. 23·6%	..
Phosphoric acid	.. ..	9·8%
Saturated acids	.. 21·2%	..
Potassium ..	.. ..	3·4%

They were separated by complete acid hydrolysis of soyabean lipositol. Saturated acids comprised 5 per cent cerebronic acid, 95 per cent of a mixture of palmitic (70 per cent) and stearic (30 per cent) acids. By partial acid hydrolysis of lipositol he obtained inositol monophosphate.

Isolation of Inositol

Lipositol was separated by him as in the case of phospholipids of nerve tissue (Folich and Woolley[5]). From lipositol inositol was separated by fractional precipitation with methyl and ethyl alcohols, followed by extraction with ether. He concluded that soyabean lipositol was very similar to brain cephalin.

Inositol from Liver Extract

Woolley[7] (1941) isolated a water-soluble, non-dialyzable fraction from liver extract, which gave positive tests for inositol. He suggested that inositol is present in liver in the form of phosphorylated ester.

Nature of Linkage between Inositol and Phospholipids

Anderson (*loc. cit.*) during his isolation of inositol from phospholipids of tubercle bacilli suggested that in the phosphatide, inositol might be linked with the carbohydrate part (as a glucoside) or with fatty acids. Woolley (*loc. cit.*) gave his idea regarding the nature of linkage between inositol and phospholipids of soyabean represented in the following lines. He stated: "Lipositol may be considered to consist of inositol with which phosphoric acid, oleic acid, a saturated acid (chiefly palmitic) and ethanolamine tartrate have been esterified and galactose has been combined in glycosidic linkage." The exact manner in which inositol is linked in the phospholipid molecule is still unknown.

Importance of Inositol

(1) Inositol is one of the constituents of Bios. It is one of the essential vitamins required by lower forms of life (except bacteria) as well as by higher animals. It is also called *mouse anti-alopecia factor* or *rat-spectacled-eye factor.*

(2) Phytin and other phosphorylated esters of inositol also function as anti-alopecia factors, though phytin is not utilized as adequately as free inositol.

(3) Its occurrence in free state as well as in ester form suggests that it might be phosphorylated in the body. As phospholipids are labile structures inside the body, free inositol or inositol monophosphate might be released from them inside the body.

(4) In the body it acts as a lipotropic factor or fat removing factor.

(5) The importance of inositol occurring in phospholipids is well stated in the following lines[2]: "The occurrence of inositol is an integral part of a phosphatide and the role of inositol in the prevention of fatty livers bring to mind the occurrence of choline in a phosphatide and its vitamin-like action in prevention of fatty livers of a different character. Perhaps the formation of lipositol is one of the uses to which dietary inositol is put by the animal."

Inositol is one of the important constituents of phospholipids, especially of human tubercle bacillus, soyabeans and nerve tissues, from which it has been isolated in pure form.

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QUININE AMBLYOPIA

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The incidence of quinine amblyopia is very very small indeed when we consider the number of patients that have to take to quinine in varying doses and over a prolonged period for the cure and prevention of malaria. Few cases are on record where it has occurred following its use as an abortifacient. Within last 8 years, I have come across only two genuine cases of quinine amblyopia. Why I say genuine, because lots of patients attribute their suddenly detected loss of vision to administration of quinine. But the ocular manifestation of toxic effects of quinine is a definite clinical entity as regards its onset, clinical course, pathological changes in the eye and visual field, which I shall deal with presently.

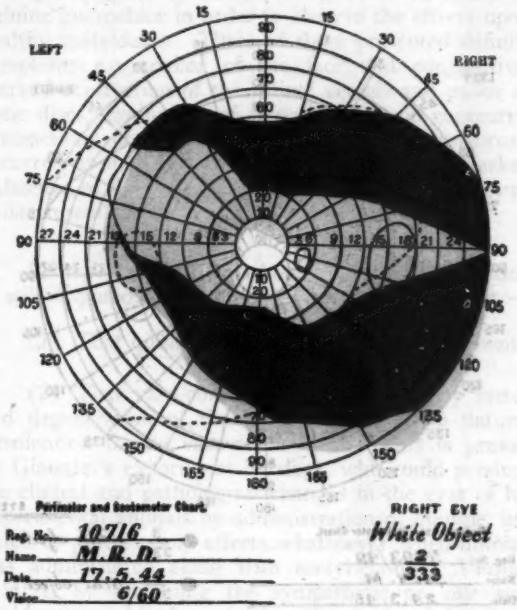
It goes without saying that the toxic effects of quinine depend more upon individual susceptibility than anything else. Apart from those rare cases where large doses of quinine are taken at a time, most of the cases are due to a small dose. I shall cite examples of these two types.

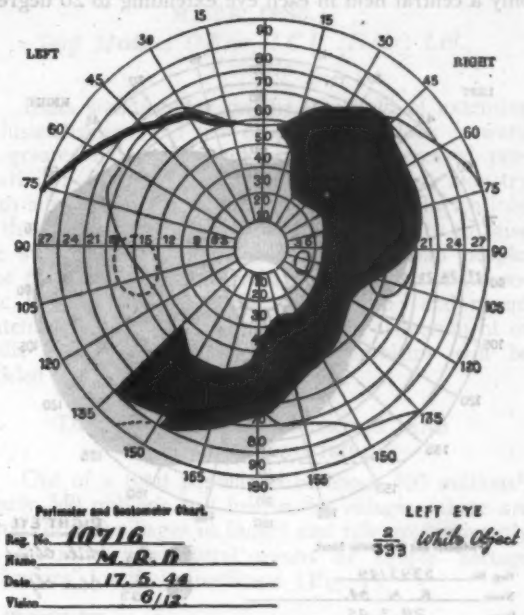
Report of the Cases

Case 1—Mrs. M.R.D., 23, Hindu female came to my consulting room on 18-11-43. She took 180 grains of quinine 8 days back and soon after she was totally unconscious. A doctor was called in and after 2 days of strenuous efforts she became conscious, but was totally blind.

When I saw her, her both pupils were widely dilated, light reflex was lost and she could not see hand movements. There was only faint perception of light on the temporal side. The fundi were pale, optic discs were pale, margins of the discs clear, arteries were very much attenuated, macula was healthy with a bright foveal reflex.

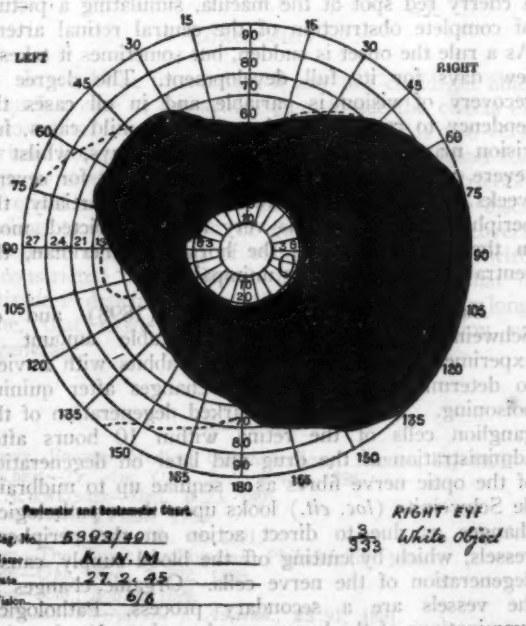
Six months after she came back to me on 17-5-44. Vision in right eye was $^{6}/_{60}$ and in left eye $^{6}/_{12}$. Field of vision was recorded with a 2 mm object at a distance of $^{1}/_{3}$ metre. In the right eye, the visual field in the horizontal meridian was within normal limits but it was constricted in the vertical meridian extending up to 25 degrees only. In the left eye the visual field was slightly irregularly constricted, more in the nasal half. The fundi of both eyes presented almost the same picture as on the previous occasion.

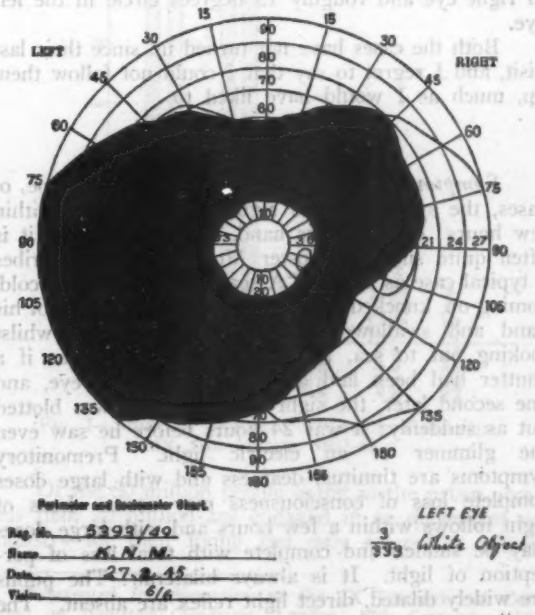




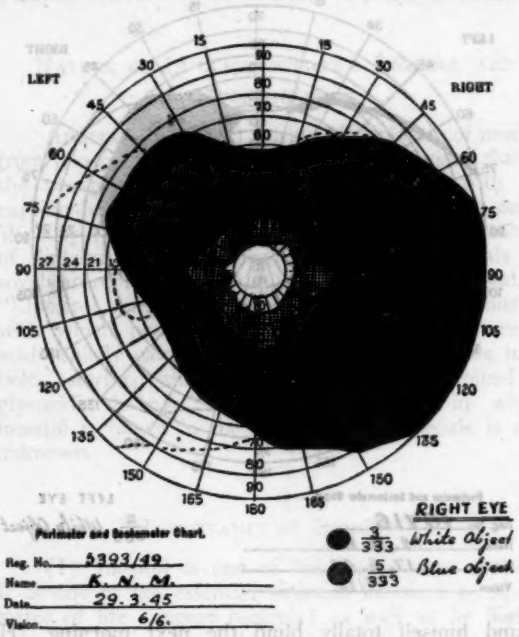
Case 2—K.N.M. 38, Hindu male came to see me on 24-2-45. He took 4 ounces of quinine sulphate mixture by mistake two and half months back and found himself totally blind the next morning. He complained of deafness but did not lose consciousness.

When I examined him his pupils were semidilated and light reflexes were very feeble. Vision was $^{6}/_{6}$

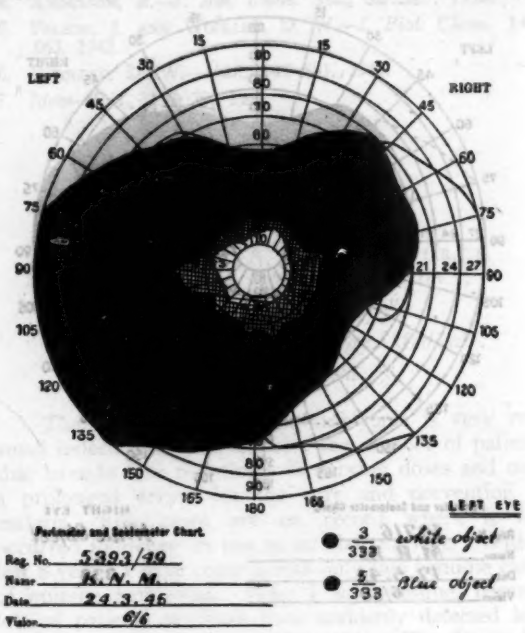




in each eye. There was slight general pallor of the discs and arteries were rather attenuated. Macula and foveal reflex were normal. The field of vision was recorded in each eye on 27-2-45 with a 3 mm white object at a distance of $^1/_3$ metre and he had only a central field in each eye extending to 20 degrees



circle. A month later on 29-3-45 his fields of vision were examined with the same object and at the same distance. The horizontal meridian showed some improvement, field of vision extending roughly to 27 degrees, but the vertical meridian remained to 20 degrees. The field for blue was done at the second



sitting with a 5 mm blue object at $^1/_3$ metre distance and it was found to be restricted to 10 degrees circle in right eye and roughly 15 degrees circle in the left eye.

Both the cases have not turned up since their last visit, and I regret to say that I could not follow them up, much as I would have liked to.

Discussion

Symptoms and signs—In the ordinary type of cases, the symptoms and sign usually develop within few hours; on the other hand, with large doses, it is often quite sudden. Foster Moore (1925) describes a typical case as such: "A sea captain feeling 'a cold' coming on, knocked some quinine on to the palm of his hand and swallowed it. Seven hours later, whilst looking out to sea, it was, he said, exactly as if a shutter had been laid down over his right eye, and one second later, the sight of the left eye was blotted out as suddenly; it was 24 hours before he saw even the glimmer of an electric light." Premonitory symptoms are tinnitus, deafness and with large doses complete loss of consciousness may occur. Loss of sight follows within a few hours and with large doses may be sudden and complete with total loss of perception of light. It is always bilateral. The pupils are widely dilated, direct light reflex are absent. The fundi are pale, the retinal arteries are very much attenuated and the discs are pale. Sometimes in the earliest stages, the retina is a bit oedematous, there is a cherry red spot at the macula, simulating a picture of complete obstruction of the central retinal artery. As a rule the onset is sudden, but sometimes it takes a few days for its full development. The degree of recovery of vision is variable and in all cases the tendency to recovery is the rule. In mild cases, full vision may be restored within a few days, whilst in severe cases the loss of vision may persist for several weeks and when it recovers its vision partially, the peripheral visual field is very much restricted. more in the vertical than in the horizontal meridian, the central vision remaining unimpaired.

Experimental work—Holden (1898) and de Schweinitz (1896) did a considerable amount of experimental work on dogs and rabbits with a view to determine the pathological changes after quinine poisoning. They found a marked degeneration of the ganglion cells of the retina within 10 hours after administration of the drug and later on degeneration of the optic nerve fibres as a sequlae up to midbrain. de Schweinitz (*loc. cit.*) looks upon these pathological changes as due to direct action on the peripheral vessels, which by cutting off the blood supply, causes degeneration of the nerve cells. Organic changes in the vessels are a secondary process. Pathological examinations of the human eye have been but few, but

have shown identical changes as in those of experimental animals.

Barabaschew (1891) experimented on six of his willing colleagues by administering 40 to 60 grains of quinine hydrochlor in order to observe the effects upon healthy individuals. Three of them presented definite symptoms *e.g.*, pallor of the face and conjunctiva, marked contraction of the retinal vessels and pallor of optic discs, diminution of visual acuity and concentric contraction of visual fields. Complete amaurosis occurred in one and lasted a very short time Marked ischaemia of the retina occurred in all cases and in two pallor resembled optic atrophy.

Pathogenesis—The pathogenesis of the condition is still disputed. Two theories are held at present:—

(1) That quinine acts directly on nerve elements.

(2) That vaso-constriction is the primary factor and degeneration of the nerve elements is a natural termination to the vaso-constriction. This is proved by Giannini's experiment on dogs, who could produce the clinical and pathological changes in the eyes of his experimental animals by administration of quinine, but failed to produce any effects whatsoever when quinine was administered along with acetyl-choline. Vermes (1905), by sectioning the sympathetic on one side could produce blindness in the other eye and thus concluded that the action of the drug is not directly on the vessel wall but acts indirectly on the vessel wall through the sympathetic.

Conclusion

It would be interesting, if any one could get hold of such a case at the acute stage, to see the effects of retro-orbital injection of acetyl-choline. It is also very curious how a patient, who has been totally blind for days or weeks and in whom extreme vasoconstriction of retinal arteries are apparent with pallor of the discs leading to degeneration of ganglionic cells, recovers a considerable amount of vision sooner or later with a constricted field. One is likely to suppose that if a tissue is deprived of its blood supply for such a long time, that tissue will not recover its function or regenerate.

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THE CONCEPTION OF INDUSTRIAL HEALTH IN INDIA—AN AWAKENING *

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India stands today on the threshold of extensive industrialization and her people rightly look forward to greater industrial employment and increased productivity. A well-planned growth of Indian industry with adequate safeguards against abnormal fluctuations in the prices of primary goods would do much to raise the standard of living of millions of Indian people. The publication of the 'Bombay Plan' (Plan of Economic Development for India, 1944, 1945) and recent statements on industrial policy by the Government of India give indication that these problems will be tackled with utmost zeal.

Distribution of Population and Principal Industries

Out of a total population of about 400 millions[1], nearly 340 millions still live in the villages (there are some 700,000 villages in India) and rely predominantly on primitive agricultural means or simple 'cottage industries' for their livelihood (Fig. 1).

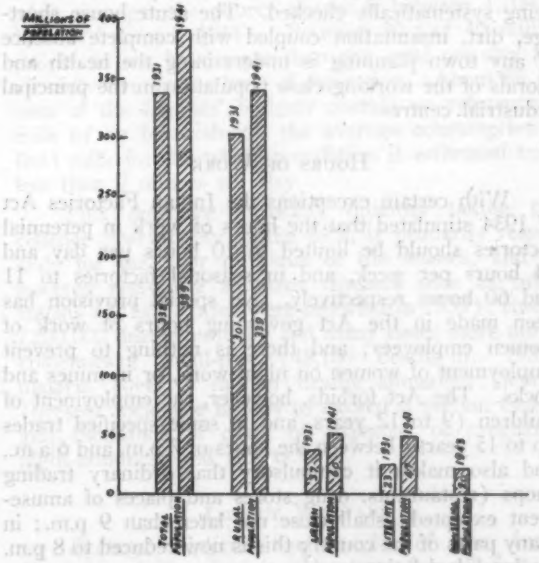

Fig. I—Showing India's Population Variation in the Decade 1931-41

Of the 50 million people living in the urban areas less than 20 million are engaged in or dependant on industry for their living and only some 2½ million

* Accepted for discussion at the IX International Congress on Industrial Medicine, London, 17th September, '48.

[1] Statistical data in this paper has been calculated on an All-India basis prior to the partition.

people (*i.e.*, 0·6 per cent of the total population) are employed in large scale industrial establishments. In 1943 for which the latest figures are available the total number of factories registered in India under the Factories Act 1934 was 13209 employing about 2,436,800 workers. The industries in India fall into two classes—organized and unorganized. An industry is regarded as organized if it employs 10,000 persons or more in establishments containing not less than 10 persons in each and includes some of the important textile mills (829,778 operatives), iron and steel factories (253,940 workers), mines (364,948 miners), railway workshops (962,010 employees), dockyards and plantations (over 1,000,000 labourers) [Table 1.]

TABLE 1—SHOWING NUMBER OF WORKERS ENGAGED IN SOME OF THE PRINCIPAL INDUSTRIES—1943

Nature of Factory	*Nos. employed*		*Remarks*
Textiles—Cotton Mills (417) ..	509,778		Present production of milled-cloth about 4800 million yards, hardly 12 yds. per head per year.
Jute Mills (107) ..	320,000	approx.	
Iron and Steel	253,940	„	Of these Tata Iron & Steel Co. alone employ over 150,000 persons.
Mines and quarries	364,948	„	Of these 135,150 worked underground and the rest in the open.
Railways	962,010	„	Indian Railways extend over 40,944 miles of which 32,967 miles are under Govt. control and the rest are owned by the Indian States and private companies.
Plantations—Tea	925,000	„	Annual production 564 million pounds of which 324 million pounds are exported, chiefly to the U.K.
Coffee	99,100	„	Annual production 310,900 cwt. of which 118,900 cwt. is exported.
Sugar refineries (150) ..	120,000	„	Annual production nearly 1½ million tons, mostly for home consumption.
Skins & hides	33,660	„	Annual production about 70 million hides, chiefly for leather for military equipment and civilian footwear.
Paper and printing	50,534	„	Chiefly paper making and publications. Newsprint requirements are all imported.
Food, drink & tobacco ..	124,736	„	

The industrial development and concentration of population is taking place in a few bee-hive industrial towns and its deleterious social consequences are not being systematically checked. The acute house shortage, dirt, insanitation coupled with complete absence of any town planning is undermining the health and morals of the working class population in the principal industrial centres.

HOURS OF WORK

With certain exceptions the Indian Factories Act of 1934 stipulated that the hours of work in perennial factories should be limited to 10 hours per day and 54 hours per week, and in seasonal factories to 11 and 60 hours respectively. No special provision has been made in the Act governing hours of work of women employees; and there is nothing to prevent employment of women on night-work, or in mines and docks. The Act forbids, however, the employment of children (9 to 12 years, and in some specified trades up to 15 years) between the hours of 7 p.m. and 6 a.m., and also makes it compulsory that ordinary trading shops (restaurants, drug stores and places of amusement excepted) shall close not later than 9 p.m.; in many parts of the country this is now reduced to 8 p.m. by local legislation.

The actual hours of working in most factories are, however, less than those prescribed by law and vary in different industries. The textile mills in almost all centres normally work a uniform 9-hour day from Monday to Friday and 4 or 5 hours on Saturday. In the coal mines the workers seldom work for more than 5 to 6 hours per day and then only for 4 or 5 days in the week; on an average, they work a 44-hour week if underground, and upto 48 hours in the open. The dockyards, larger engineering plants and almost all railway workshops generally work a 48-hour week.

There is much to be said in favour of shorter hours of work in the Indian factories. Climatic conditions for the major part of the year make labour particularly irksome in India. The air inside a factory is often hot, stagnant and full of dirt and dust and speaks of the absence of modern methods of ventilating devices. Under the rigours of Indian climate manual labour is not possible for any length of time and consequently in most factories absenteeism is very high, and production rate very low. In the jute mills of Bengal, for example, the number of working days lost per month is some times as much as a week on an average for all workers. In the Western countries partly due to rationalization and partly to shorter hours of work industrial output has been greatly increased and unemployment markedly decreased.

TABLE 2—AVERAGE DAILY WAGES IN SOME OF THE PRINCIPAL INDUSTRIES—1941

Occupation (Men over 21)	*Daily wages for 10-hour day*	*Average daily wages in Britain for 8½-hour day*
	Rs. A. P.	*s. d.*
Agricultural labourers	0 2 3 (3*d.*)	10 0
Tea-garden labourers	0 5 0 (5½*d.*)	.. ..
Miners—		
Underground ..	0 9 0 (10*d.*)	16 6
Open-workings ..	0 7 6 (8½*d.*)	15 0
Unskilled workers in industry ..	0 7 1 (7½*d.*)	12 0
Skilled workers in urban industries such as textile mills, engineering workshops etc. ..	1 5 0 (1*s.* 11*d.*)	17 0

Average Income

A worker whether on land or in industry receives very low wages (Tables 2 & 3).

Table 3—Average Basic Wages per Month (All Industries)—1945

Worker	*Monthly basic wages*
Adult Male ..	Rs. 18 to Rs. 35 (£1 7 0 to £2 12 6)
Adult Female ..	Rs. 12 to Rs. 25 (£0 18 0 to £1 17 6)
Children ..	Rs. 7 to Rs. 12 (£0 10 6 to £0 18 0)

A tea planter may earn 4 as. to 5 as. per day (4½ to 5½d.) and a skilled worker in a textile mill or engineering workshop upto Rs. 1/5/- (1s. 11d.).* Women workers are paid at a much lower scale, about half that of men, for the same number of hours and even for equal work. This disparity of basic rates of pay for men and women for the same job is a crying economic injustice which ought to be remedied. Children's wages in India are unbelievably low and thousands of them are often employed in sedentary occupations for only 1 anna or 2 as. (1 or 2d.) a day, sometimes working for the same number of hours as the adults.

Since 1942, due to war time exigencies, the earning capacity of an worker had improved considerably, and taking an average of all industries an adult worker today receives a basic pay of Rs. 18-0-0 to Rs. 35-0-0 (£1-7-0 to £2-12-6) per month. To this must be added one supplementary income, introduced during the war to meet the high cost of living, and still continuing, known as the Dearness Allowance. At present it varies from 30 per cent to 70 per cent in different provinces and the minimum dearness allowance paid to the lowest grades of wage-earners is generally Rs. 30/- to Rs. 40/- per month in order to make up the pay-packet to a minimum of Rs. 55/- to Rs. 65/- (£4-2-6 to £4-17-6), according to the cost of Living Index in the particular area (Table 4).

Table 4—Working Class Cost of Living Index

Year	*Cost of living index in the industrial cities in India* Base—1934=100	*U.K.* Base—1939=100
1939	106	102
1940	109	119
1941	122	128
1942	160	129
1943	227	128
1944	254	130
1945	260	131
1946	267	131
1947	280	..

Nutrition and Deficiency Diseases

After meeting the cost of shelter and clothing from their meagre earnings which takes away about a quarter of their income, Indian workers cannot afford a well-balanced diet. Aykroyd (1937) in his survey of nutritional problems in India showed that in pre-war days the cost of obtaining the minimum subsistence diet, inadequate and ill-balanced, consisting of cereals and very little else, came to at least Rs. 2/8/- a month (4s. 3d.); a reasonably balanced diet, judging by Indian standards, cost between Rs. 5/- and Rs. 6/- a month (7s. 6d. to 9s.). It was further estimated that in normal times about 30 per cent of the population were quantitatively underfed, and at all times the quality of the diet suffers more than the quantity (Fig. 2).

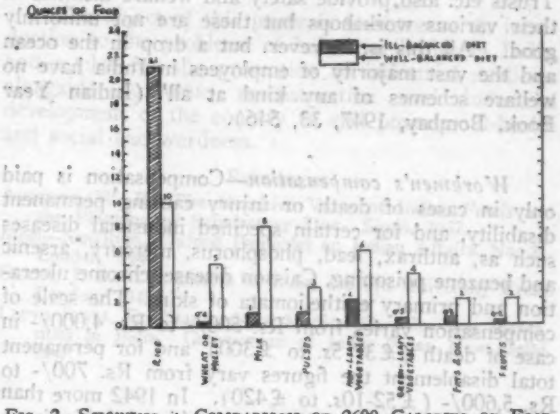

Fig. 2—Showing a Comparison of 2600 Calories of Foodstuffs in India as Obtained at Present in an *ill-balanced diet*, and which could be Obtained in an *well-balanced diet*, at Approximately same or Slightly Higher Cost.

The average daily caloric intake for a working adult amongst the poorer classes is between 1,700 to 1,800 (Aykroyd, 1944). Between 30 and 40 million people do not have more than one meal per day and virtually live on the level of starvation. About 88 per cent of the families' budgets contain no reference to milk or its by-products; the average consumption of fluid milk for the whole population is estimated to be less than 2 ounces per day.

This defective nutrition, quantity taking precedence over quality of the food, is primarily responsible for the poor physique of the masses, and the lowered resistance to many diseases such as tuberculosis (341,659 cases were reported in 1939); cholera (200,000 deaths annually and many times more during epidemics): small-pox (48,104 deaths in 1939); malaria (over 1 million deaths annually); leprosy (present incidence known to exceed 2 million cases); and thousands of cases of anaemias of pregnancy, night-blindness and rickets.*

Labour Welfare

Factory Welfare—Section 33 of the Indian Factories Act makes it obligatory for all factories employing over 150 workers to provide adequate shelters for the use of workers during periods of rest. Apart from this a number of modern industrial establishments in India do provide 'tiffin rooms' and rest

*All monetary calculations in this paper are taken as Re 1 equivalent to 1*s. 6d.*

*Probably there are many more cases than recorded for data are always obtained from hospitals and dispensaries, while many suffer and die in silence in the village.

shelters for their work-people and many of them have also established 'tea stalls' on the premises. There has been very little effort made, so far, to run co-operative canteens on lines such as are known in Britain, and other European countries.

Many labour-employing organizations, such as the Tata Iron Works, Textile Mills, Railways, Port Trusts etc. also provide safety and welfare facilities in their various workshops but these are not uniformly good. 'All this is, however, but a drop in the ocean and the vast majority of employees in India have no welfare schemes of any kind at all' (Indian Year Book, Bombay, 1947, 33, 546).

Workmen's compensation—Compensation is paid only in cases of death or injury causing permanent disability, and for certain specified industrial diseases such as, anthrax, lead, phosphorus, mercury, arsenic and benzene poisoning, Caisson disease, chrome ulceration and primary epitheliomata of skin. The scale of compensation varies from Rs. 500/- to Rs. 4,000/- in case of death (£37 5*s*. to £300), and for permanent total disablement the figures vary from Rs. 700/- to Rs. 5,600/- (£52-10*s*. to £420). In 1942 more than Rs. 18,69, 360 (£140,202) was paid out in India as Workmens' Compensation for some 44,443 cases of accidents and occupational diseases, averaging Rs. 42/- per case.

Trade Unions—Since the first world war rapid progress has been made by the trade union movement, led by many honest and genuine leaders, and today, with a membership of over 600,000, the organization has a strong representation both at the centre and in the International Labour Organization.

Many trade unions, apart from securing concessions from employers and settling grievances, also provide a variety of welfare schemes in the form of dispensaries, facilities for education and recreation, co-operative stores and cheap grainshops for the benefit of their members.

Present Health Standards

The standard of health in India is low. Both the birth-rate and crude death-rate are approximately double that of Britain. The high birth-rate, however, is to a very large extent nuetralized by heavy mortality both of infants and mothers at child-birth (Figs. 3 & 4).

Grant (1943) estimated that the mean-age in India was 23·2 years as against 30·6 in England and Wales, and the expectation of life at birth was about 27 years in contrast to an expectation of 67 years in Australia, of 63 years in Great Britain and Germany, and of 47 years in Japan.

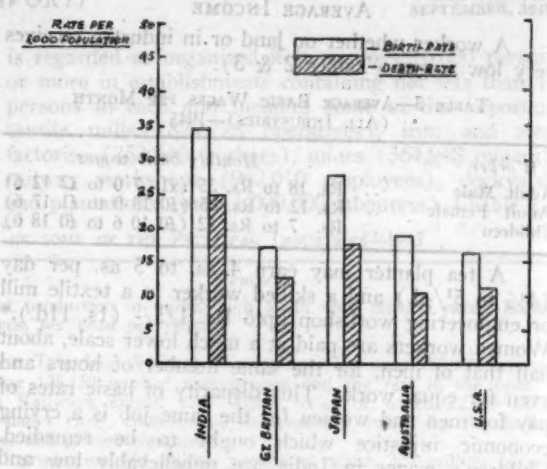

Fig. 3—Showing Some National Birth-rates and Crude Death-rates per 1,000 Population (1939)

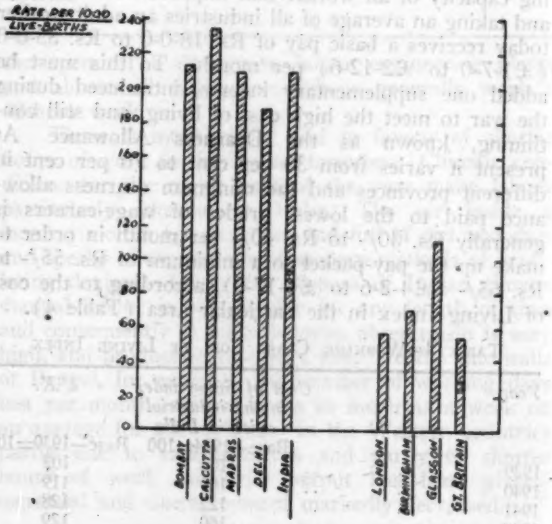

Fig. 4—Showing Infant Mortality in Some Great Cities (1941)

Statistics of health and mortality which are collected and published in India relate to the whole community, and with the sole exception of accidents in factories (Table 5), no statistics are compiled separately for industrial workers alone.

In the absence of necessary data it is not possible to generalize on matters of industrial health, sickness and fatigue, or occupational poisoning and dermatoses etc.

TABLE V—STATISTICS OF ACCIDENTS IN FACTORIES (EXCLUDING MINES)—1942*

Type of accident	Perennial factories	Seasonal factories	Total for the year
(1) Fatal	276	47	323
(2) Serious (causing permanent disability)	8,799	312	9,111
(3) Mild (causing enforced absence from work for over 48 hours) ..	43,790	950	44,740
Total			54,174

* Corresponding figures for the mines in 1945 were:

Fatal accidents	307
Serious accidents	1,478
Cases of minor injuries	8,724
Total	10,509

The frequency of accidents in the Indian factories is high; about 1 in 40·6 workers sustain physical injury in course of his work, and 1 in 6,818 meets with his death, that is to say, the annual accident rate is about 2,463 per 100,000 employees, and the accident mortality or fatality rate is 6 per 1,000 reported cases of accidents. Recently a random collection of 500 samples of blood amongst the tea plantation workers showed an average hæmoglobin content less than 50 per cent. Malaria, kala-azar, intestinal disorders and hookworm infections are rampant (Lloyd Jones, 1947). In 1945, the malaria morbidity rate in the mining settlements was estimated to be 45·08 per 1,000 colliery workers but no mention is made of a single incidence of pneumonokoniosis (Annual Report of the Chief Inspector of Mines, 1945). There is no reason to suppose that the Indian miners do not suffer from anthracosis and silicosis or the cotton mill operatives from byssinosis; it only shows lack of any investigation in these directions.

Social services in the factories, and in the country generally, are very meagre. There are some 42,000 doctors and only 7,000 qualified nurses in a country of 400 million people, averaging roughly 1 doctor to every 9,500 of population (Great Britain 1 to 776), and 1 nurse to nearly 57,000 (Great Britain 1 to 435). Three-quarters of the municipalities in India are still without qualified health officers of any kind. There is no nation-wide provision for sickness, unemployment or old-age benefits. There is no Poor Law, and the lot of the worker at present is one of debt and destitution.

But things are taking a turn for the better. Both the Government and the people are alive to these problems and there are indications that these deficiencies will be tackled with utmost zeal. This is evidenced from the various legislations now in the process of preparation such as the Industrial Injuries Act and the Employees State Insurance Act etc. Thus, it may be looked forward to, with confidence, that through conscientious "team-work" between the State, the employer, and the worker the industries in India will prosper and her people will soon attain and maintain a worthy standard of living.

CONCLUSION

The object of this paper is to serve as an introduction to the problems of industrial health in India, and a plea for the promotion of industrial medicine alongside economic reconstruction and industrial development of the country to end poverty, ill-health and social backwardness.

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TROPICAL EOSINOPHILIA

(A manifestation of Malaria ?)

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Synonyms—Idiopathic pulmonary eosinophiliaemia, eosinophilic pseudo leukaemia, pseudo tuberculosis with eosinophilia.

History—Considerable interest has been aroused in this country since the publication in 1940 by Frimodt-Moller and Barton of 175 cases with a symptom complex characterised by long chronic duration and more or less benign course with low fever, a very nasty cough, mostly spasmodic in character, scanty or no expectoration, occasional haemoptysis, loss of weight, marked relative and absolute eosinophilia, characteristic x-ray appearances and a dramatic response to arsenotherapy.

Geographical distribution—As the name of the disease itself suggests, it is clearly a disease of the tropical countries. Most of the reports of this disease are from the coastal towns and the neighbouring villages. But according to my opinion, as well as per all the reports that I have come across, almost all the cases have occurred in places where malaria is prevalent either in endemic or hyperendemic form.

AETIOLOGY

The real cause of this interesting condition has not yet been found out and hence, many views have been put forth regarding its causation. There is no doubt that eosinophile calls play a definite role in the immunity mechanism of the body although much work remains to be done to elucidate all facts in relation

* A paper read at the clinical meeting of the Bhavnagar Branch of the Indian Medical Association held on the 1st Feb., 1947.

to its role. It is a well known fact that eosinophilic count is high in allergic states produced by amœbiasis, ascariasis, filariasis, asthma and other parasitic diseases, but high eosinophilia with pulmonary shadows was first observed by Loffler and described as "Loffler's syndrome". But it is proved that tropical eosinophilia is different from Loffler's syndrome in which there is transient eosinophilia with tendency towards spontaneous recovery.

Frimodt-Moller believed it to be allergic in nature. But most other writers do not consider allergy to be the determining factor.

Another theory is of its being due to virus infection.

A third possibility is that of its being due to some organic infection. But, so far attempts to discover the organisms from sputum, culture and various other agglutination tests have failed.

Personally, I attribute this condition directly or indirectly to malarial infection.

What actually led me to the idea of its being due to malarial infection was a very intelligent patient aged 60 years, who came to me for treatment two years back. His only complaint was a sense of breathlessness, all the time, especially on slight exertion. He had two N.A.B. injections from some other doctor, I got his blood examined which revealed 48 per cent eosinophilia and a very heavy infection with malarial parasites.

Reasons for my belief are many.

Almost all the cases that I have come across were infected with malaria. It is not absolutely necessary that the patient must have the typical classical picture of malaria. But blood examination in the majority of cases revealed M.P. present at one stage or another. Eosinophilia, I believe, is an attempt on the part of Mother Nature by means of immunity mechanism for spontaneous recovery from malaria infection. Just as Black Water Fever is a drastic attempt on the part of Nature towards spontaneous recovery from malaria, similarly, I believe, eosinophilia is a slow chronic but benign attempt on the part of Nature towards spontaneous recovery from malaria.

There are many other points which I am describing below for tropical eosinophilia to be considered as having some direct or indirect concern with malarial infection.

(1) Malaria is a tropical disease. Similarly, tropical eosinophilia is found exclusively in the tropics only.

(2) All the reports of tropical eosinophilia are from areas where malaria is an endemic or hyperendemic disease.

(3) Eosinophilic response in the blood is mostly due to parasitic disease and malaria is also a parasitic disease. The other parasitic infections are in the tissue or different organs of the body but not direct in the blood. But, malarial parasites are found directly in the blood. A toxin produced by a parasitic disease of some specific organ or tissue is not much in quantity, at the same time, it can not have such a free and direct access to the blood circulation as happens in malaria. Malaria is a direct blood cell infection. Its toxins produced are greater in amount and has a direct and free access to the blood circulation and so, eosinophilic response is much in excess than other parasitic diseases. It may, here, be asked why eosinophilia does not occur in all patients of malaria. Many of my professional brothers must have observed urticarial reaction in patients suffering from malaria. Urticarial reaction does not occur in all cases of malaria. So, might be the case of eosinophilia. It is possible that malaria infection might be producing various types of toxins. A particular type of toxin might produce urticaria while another type of toxin might produce eosinophilia.

(4) It has been observed by me and I have seen many reports in different medical journals that in 50 per cent of the cases of tropical eosinophilia, there is an associated enlargement of the spleen. It is a well known fact that spleen is the only organ in the body which gives us a definite clue as to whether a patient has suffered from malaria. This associated enlargement of spleen in cases of tropical eosinophilia, I consider, a very important point, in favour of my view of tropical eosinophilia being a manifestation of malaria.

(5) I tried injections of quinine alone in cases of tropical eosinophilia. In many cases, there was as dramatic a response from quinine as is observed with arsenic injections.

(6) And last but not the least, is the response of tropical eosinophilia to arsenotherapy. It is quite true that arsenic is the sheet anchor in the treatment of chronic, resistant and relapsing malaria. At the same time, arsenic is the specific remedy in many of the protozoal diseases, *e.g.* Syphilis, Rat-bite fever, amœbiasis, etc.

In short, from the geographical distribution, its prevalence mostly in malarious districts and from clinical as well as therapeutic viewpoints, I think, tropical eosinophilia can be considered to be a disease directly or indirectly attributable to malarial infection.

I would just like to mention here the case of a medical practitioner. His blood report showed a high eosinophilic count .At the same time, there were many malarial parasites in his blood. Ring forms, free merozites as well as malarial pigments were present in his blood. It may be noted here that malaria pigments are the last things to disappear from the blood of patients of malaria, though, apparently, the patient had recovered from the attack, since very long.

The medical practitioner, above referred to, was quite robust, hale and hearty and he never had reason to believe that his blood might contain M.P. and yet they were there on repeated blood examinations.

Predisposing factors—In most of the patients, a history of low irregular fever is available. Exposure to dust and cold or an attack of influenza starts the trouble.

Sex—Apparently, it seems that the incidence of the disease is more among men than in women. An analysis of my 19 cases during the last four months, revealed, that 8 were females and 11 were males.

Age—It is very common between the age of 20 and 50. The youngest patient that I had seen, last year, was a girl of 9 years.

Season—The disease is met with any time of the year but more patients are observed during and after the winter months. It may be noted that malaria is also very common during these months. I have particularly noted that a great number of patients of tropical eosinophilia turn up at the time when malaria is declining. These are the patients who have suffered from malaria but were either not treated at all or partially treated.

Symptoms

An infinite variety of symptoms are observed.

The commonest and most troublesome symptom is *cough*. It is very distressing and mostly spasmodic in character. During day time there is comparatively little trouble but it becomes worse at night. It even wakes up the patient from deep sleep. There is no expectoration at all or at the most a very little frothy mucus is brought out. Patient feels breathlessness, sometimes, actually amounting to an asthma-like attack but true typical asthma is never observed.

In the majority of patients, an insidious onset is observed, with low temperature and troublesome cough which gets worse as time passes.

Many patients relate that the onset was like an attack of coryza or a mild influenza, which later became chronic.

Sputum never becomes purulent or offensive in odour. Rusty or prune juice sputum is never observed. With paroxysms of cough, however, blood streaks or even a small amount of blood is expectorated. Frank hæmoptysis even may occur.

Three years ago, I had a patient, a young man of twenty-three. His only complaint was a very distressing paroxysmal cough, bringing a teaspoonful of blood at the end of the paroxysm. He had no fever but there was marked congestion of the throat. Apparently, it looked to be a case of pharyngitis or severe tracheitis. All sorts of treatment were given to him for one full month but with no effect. Ultimately, I gave him arsenic injections which completely relieved him of his trouble.

Some cases seem to have an acute onset with fever going up to 101°F or 103°F, followed a few days later by troublesome cough.

Appetite of these patients is very poor and loss of weight is also complained of. At the same time, because of the troublesome cough and dyspnœa, patient is even afraid of eating; consequently, loss of weight is considerable.

Because of low range fever, loss of weight and appetite, associated with cough, majority of patients are labelled as suffering from pulmonary tuberculosis.

Pain in the chest, localised or generalised, is sometimes complained of.

Various types of gastro-intestinal troubles are also complained of.

Signs

Physical Examination—Inspection:—chest bilaterally symmetrical with equal movements on both sides.

Vocal fremitus:—Unaltered.

Percussion note:—Tends to be hyperresonant.

Auscultation:—Reveals harsh breath sounds with prolonged expiration. An evidence of mild bronchospasm with bronchitis with fine crepitations all over the chest or sibilant and sonorous rhonchi are also heard. Signs of congestion and consolidation are occasionally observed in some cases.

The patient is invariably anæmic. In 50 per cent of cases, enlargement of spleen is found.

Laboratory findings—(1) Blood examination reveals an increase in total leucocytes which may range from 13,000 to 50,000. Eosinophile percentage may vary from 20 to 50. An eosinophile count of 10, or 15 per cent is taken as normal by most of the authors. It is very essential that blood must be repeatedly examined for M.P. and malarial pigments. Even if malarial pigments are found, it is a positive proof that the patient has suffered from malaria.

The physical signs and the mildness or severity of the disease has no direct bearing with the percentage of eosinophile cells.

Wassermann Reaction is positive in this condition while Kahn's test is negative.

(2) Sputum examination reveals a great number of eosinophile cells, fair number of lymphocytes and epithelial cells.

Acid-fast bacilli are always absent except when there is an associated infection of the same in the lungs.

X-ray findings—X-ray examination of the chest invariably reveals lesions of one type or the other in the lungs. The lesions are not always completely revealed by screening alone.

The skiagram reveals a diffuse mottling of both the lungs. The lesions are bilateral and symmetrical. They are particularly marked at the hilar regions and bases. An average single focus is about the size of a pin head with a central dense zone and comparatively light, ill defined and blurred outline. The mottling is more of a coarse nature than that of miliary tuberculosis, in which there is no distinction between central and peripheral zone. Occasionally, instead of discrete mottled shadows, linear markings of increased density radiating from the hilum of fan-shaped streakings are observed.

Diagnosis

This is not at all difficult if the condition and its various manifestations are borne in mind.

It is just possible that one may deliver a very fine lecture on this disease but may fail to diagnose it when actually the case is before him if the condition is not in his mind.

The outstanding symptom of cough which is worse at night with a paroxysm and asthma-like attack, with low fever and progressive loss of weight mostly leads a medical man to suspect the disease.

When cough is the only outstanding symptom in the early stages, I insist on the question whether at the end of the paroxysmal cough, he feels a sense of breathlessness or a sort of heavy breathing.

This sort of difficult breathing is not found in any condition except tropical eosinophilia. I diagnose most of my patients from this symptom alone.

Only a fortnight back, an acquaintance of mine consulted me for his troublesome cough and low grade fever of one month's duration for which, his sputum was examined and X-ray taken at the instance of another doctor. It was probably a difficult case to be diagnosed only from symptoms and physical findings but I inquired of him about the heavy breathing after the paroxysm of cough and that led me to it being due to tropical eosinophilia. I gave him two N.A.B. injections and almost all his symptoms vanished after that.

Splenomegaly, if present, would be of additional diagnostic significance.

Many such cases are labelled as tuberculosis and this is quite understandable from the similarity of signs and symptoms and X-ray appearances. I have come to a definite conclusion that it is always wise to examine the blood of such patients with tuberculosis like symptoms. Not only it will lead us to the right diagnosis but it will save a lot of worry and anxiety to the patient and his relatives.

The other conditions to be differentiated are asthmatic bronchitis and eosinophilia with pulmonary shadows.

In the typical bronchial asthma, high leucocytic count is rarely encountered and eosinophile percentage is not very high. Moreover, the paroxysms of dyspnoea are very severe with much anxiety, restlessness and a sense of suffocation.

The allergic bronchitis resulting from inhalation of dust, fungi or other allergens can also produce eosinophilia upto 10 or 20 per cent and differential diagnosis may then be difficult. A history of allergy in family, rhinitis and other allergic manifestations in the patient should be taken into consideration.

Loffler's syndrome is distinguished by being a mild transient eosinophilia without splenomegaly with fluctuating pulmonary shadows shown by skiagram and having a tendency to spontaneous recovery.

Prognosis and Course

Prognosis is extremely good. If recognised and treated effectively, recovery is very rapid. But if untreated and left to itself, there does occur a spontaneous recovery after a very prolonged and troubled time, if patient has got very good vitality, otherwise, other diseases, *e.g.* tuberculosis and secondary anaemia may supervene.

Relapses—Relapses are not uncommon. I have noted five cases of relapse, three of thm were my own cases. Relapses however do not occur earlier than two years. The real cause for a relapse is difficult to find but cases noted by me were probably due to insufficient treatment.

All the cases of relapse were living in the same environmental conditions as before and all had suffered from malaria. Perhaps, the relapse may be attributed to a sort of sensitisation to a particular type of malarial toxin.

Eosinophilia during Pregnancy—I had the opportunity to treat two such cases during pregnancy. One case was running the sixth month of gestation. I treated her with Acetylarsan injections. She was completely relieved after a course of six injections but unluckily, she had placenta praevia and she aborted at the seventh month but otherwise she had no other trouble. The other case that I came across was in the ninth month of pregnancy. I had treated her with original Bayer's Neo-Salvarsan. A course of only two injections was administered. She delivered a still born girl at full term. Whether the still birth was due to eosinophilic condition or extreme anæmia of the patient or due to arsenic injections is difficult to say.

Treatment

Various treatments have been tried but arsenic has been found to have a specific effect on this malady.

Intravenous injections of arsenic work like a charm. Patients get immediate relief. Even one single injection of 0·45 gm. of N.A.B. changes the whole outlook of the patients

To have complete cure, there must be adequate treatment. But how much of arsenic can be called adequate is difficult to say.

I usually start either with 0·15 gm. or 0·3 gm. of N.A.B. and then 5 injections of 0·45 gm. are given. I never like to go beyond 0·45 gm. The higher doses can be given but majority of our Indian patients do not tolerate doses higher than 0·6 gm. It is always safe and at the same time, there is no reason to hurry with the treatment. Intravenous injections of arsenical preparations must never be repeated before one complete week.

If the intramuscular route is to be preferred, Acetylarsan or Metarsenobillon are the drugs of choice. I have tried both and they work equally well as intravenous N.A.B.

Injections of Soamin also work well. On the whole, twenty grains of soamin must be parenterally administered.

After the first injection of arsenic, there is a possibility of reaction, manifested by a rise of temperature even upto 103°F. It usually goes down in a single day but may persist even longer. The reaction is particularly severe in patients who have M.P. in their blood. It is a well known fact that arsenic works like a provocative in malarial infection. It is wise to give quinine injections in such cases after parenteral administration of arsenic.

I have tried quinine alone in many cases, particularly in the cases where there is heavy infection by malarial parasite. It works equally well like arsenic.

After six injections, the total white cell count falls down to 10,000 to 15,000 with eosinophile percentage ranging between 10 and 20. Compete restoration of white cell count and eosinophile percentage to normal has not been observed in any case, but no subjective symptoms are encountered. The increase in weight is steady, clinical and radiological signs clear up and splenomegaly gradually disappears.

Summary

(1) A review of 19 cases of tropical eosinophilia treated during four months is given.

(2) Blood examination of patients suffering from tropical eosinophilia revealed—

(*a*) an increase of eosinophile cells ranging from 20 to 86 per cent.

(*b*) a total increase of white blood cells.

(*c*) Presence of M.P. or malarial pigments at one stage of the examination of the blood or the other in most of the cases.

(3) Out of 19 patients treated, 17 were local (*i.e.*, residing in Bhavnagar, where malaria is an endemic disease), one case was from Indore and one from Jharia. Both the latter cases had M.P. in their blood.

(4) Geographical distribution of the disease *i.e.*, tropics, presence of M.P. in the blood and therapeutic response to quinine and arsenic had led me to believe that the disease has got some direct or indirect relation to the malarial infection.

(5) It is either an immunity mechanism by the organism towards spontaneous recovery from malarial infection or an allergic reaction due to some peculiar malarial toxin.

Acknowledgment

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B. C. G. VACCINE—ITS EFFICACY AND DISADVANTAGES

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Disergarh (Burdwan)

Efficacy

In order to assess the efficacy of B.C.G. a review may be made of the more important and conclusive investigations and trials that have been carried out in various countries. As experimental demonstration of immunity in laboratory animals is notoriously difficult and since it has no close bearing on human tuberculosis, it has not been included in this discussion.

(1) *Hyge's work in a girls school at Copenhagen*—Hyge's work furnishes the best evidence hitherto advanced for the efficacy of B.C.G. vaccination. Although it deals with a number of tuberculosis cases at a school, it seems as if well-planned experiment was performed on hundreds of laboratory animals.

Hyge reported an epidemic of tuberculosis in a school for girls between 12 and 18 years of age. Before exposure 105 were tuberculin positive, 106 were vaccinated with B.C.G. and 94 were tuberculin negative. The source of infection came from a temporary teacher who worked in the school for a short time. The duration of exposure was 2 months in all these groups. They were followed up during the first three years after exposure and the result is as follows:

Group	*No. of pupils*	*No of cases of pulmonary tuberculosis*	*Morbidity rate per cent.*	*Mortality*
Tuberculin (+)ve ..	105	2	1·9	Nil
B. C. G. ..	106	2	1·9	Nil
Tuberculin (—)ve ..	94	41	43·6	1

Hyge's work shows in unmistakable colours how effective was B.C.G. in prophylaxis of pulmonary tuberculosis among the school girls.

(2) *Wallgren's investigation at Gothenburgh, Sweden*—Wallgren 1934 vaccinated every infant that was considered to be in danger of infection from family contact. A striking result was obtained. The infantile tuberculosis death rate fell from 3·9 per thousand in 1927, the year before the vaccination was introduced to 0·3 per thousand in 1933—a fall of 92%. As other factors like segregation of new born infants were present, Wallgren does not claim the entire improvement to be due to B.C.G. vaccination. He later on (1948) states in response to Wilson's (1947) criticism to B.C.G.," although many children were isolated until three months of age before being subject to exposure, after this period they nevertheless were exposed to virulent infection without falling ill from pulmonary tuberculosis, save a single case. This is a result which surely the whole battery of prophylactic measures known to us cannot parallel when dealing with children of this age that have not been vaccinated before exposure

to infection. Further experience has shown me that cases of clinical tuberculosis, occurring after an adequately performed B.C.G. vaccination are extremely rare and remarkably benign. It is also remarkable that I have not seen a single adequately vaccinated child die from tuberculosis after having been infected, nor have I seen a single case of tubercular meningitis among vaccinated children."

(3) *Heimbeck's Investigation on Nurses*—Heimbeck (1936) carried out this investigation on probationer nurses entering Ulleval Hospital at Oslo during the years 1924 to 1935. Every nurse was subjected to Von Pirquet test on entry. This was repeated periodically on those who failed to react. From 1927 onwards, all negative reactors were vaccinated subcutaneously with B.C.G., as a result of which more than two thirds became Von Pirquet positive. The subsequent follow-up revealed results given in the table above:

Group	*Sub-group*	*No. of nurses*	*Nurse Observation years*	*No. of pulmonary tuberculosis*	*Rate per thousand Obs. years*
Von Pirquet (+) on entry		625	2659	4	1·5
Von Pirquet (—) on entry	Not vaccinated	280	561	13	23·2
	Vaccinated & became (+)ve	287	910	1	1·1
	Vaccinated & remained (—)ve	107	204	1	4·9

The above way of analysis was open to criticism since the initially tuberculin negative cases were transferred to positive group within a year of primary infection.

Heimbeck in 1947 published the result of vaccination of nurses during their three years' training. Each nurse remained in the same group in which she started at the entry. These nurses lived exactly under the same conditions and all were exposed to the same amount of infection. The result can be briefly summarised as follows—

Group	*Morbidity per thousand Obs. years*	*Mortality per thousand Obs. years*
B. C. G. vaccinated ..	24·1	2·1
Unvaccinated tuberculin (—)ve	141·2	14·6

Heimbeck, in 1948, published his results of observation on nurses for another 10 years. The result is based on person years of observation. It is seen that during the training period, the morbidity in the tuberculin negative non-vaccinated group is nearly 12 times higher than in the tuberculin positive group and about six times higher than the vaccinated group.

(4) *Chicago Investigation*—This was carried out by Rosenthal, Blahd and Leslie (1945) at the Cook County Hospital in Chicago. The vaccine was given to every alternate infant whose mothers consented at the age of 3 to 7 days by multipuncture method, the other infants serving as controls. Infants were also divided into two groups having contact or having no contact with known cases of tuberculosis. The subsequent follow-up reveals the following:—

Group	*Sub-group*	*No. of children*	*No. of cases of tuberculosis*	*No. of deaths from tuberculosis*
Non-contact	Vaccinated	1204	3	1
	Control	1213	23	4
Contact	Vaccinated	98	1	0
	Control	63	4	3

The table shows quite clearly that morbidity and mortality of tuberculosis is considerably lower in the control than in the vaccinated group.

(5) *Use of B.C.G. in Denmark*—Holm reports that since the year 1940 B.C.G. vaccination has been adopted as an essential weapon in the control of tuberculosis in Denmark. In that country, vaccination is mainly used in tuberculosis environments. All tuberculin negative children in a tuberculous environment as well as the new-born are vaccinated. On development of tuberculin sensitivity, they are allowed to remain in the environment. Since the introduction of vaccine the mortality from tuberculosis among the children in tuberculous environment have been reduced to almost zero, though previously mortality from tuberculosis among such children was very high. Those who suffer have a much milder form of tuberculosis than previously observed. Holm concludes that B.C.G. vaccination gives considerable though not absolute protection against tuberculosis.

(6) *B.C.G. Vaccination in Oerobro, Sweden.*—This has been carried out by Malmros (1948) in the district of Oerobro which has a population of 2,30,000. Practically all the new-born are vaccinated. The whole population is systematically and successively tuberculin tested and the non-reactors are vaccinated if they give their consent. During the period 1942-47, 22,413 persons have been vaccinated of whom 8,040 are new-born; 7,836 children over 1 year of age and 6,537 adults. Of these vaccinated cases only one case of tuberculosis developed in a probationer nurse who was tuberculin negative in spite of two B.C.G. vaccinations.

(7) *Vaccination of more susceptible race with B.C.G.*—Aranson and Palmer (1946) carried out this investigation among the North American Indians who are a more susceptible race with a high tuberculosis death rate. The subjects ranged from 1 to 20 years of age. Every alternate non-reactor was vaccinated intracutaneously with B.C.G., while the other alternates comprising the control were injected with sterile saline. The two groups were observed for six years during which period about 20 per cent of each group was exposed to infection. The result has been tabulated.

Tuberculosis mortality is here seen to be about seven times higher in the control than in the vaccinanted group. Such a result can however hardly be expected from populations with a higher natural resistance. The tabulation is as follows:

Group	No. of subjects	No. developing non-pulmonary tuberculosis	No. developing pulmonary tuberculosis	Tuberculosis case rate per thousand person years	No. of deaths from tuberculosis
Vaccinated	1550	9	8	2·0	4
Non-vaccinated	1457	48	20	9·0	28

(8) *New York City Investigation*—This was done on children in tuberculous families. Practically all children were under one year of age. An infant over one month was not considered unless it was both tuberculin and x-ray negative. In the first two years, the vaccine was given by mouth and after that by intracutaneous method. Between 1926 and 1932, children were divided into two groups according to whether the mother wished her baby to be vaccinated or not. In 1933 and 1934 this system of grouping was altered. Only the infants whose mothers consented were selected. Every alternate of such babies was vaccinated and the other alternates served as controls. Levine and Sachett have given the results as follows:—

Period	Vaccinated Group No. of children	Vaccinated Group No. of deaths from tuberculosis	Vaccinated Group Tuberculosis mortality %	Control Group No. of children	Control Group No. of deaths from tuberculosis	Control Group Tuberculosis mortality %
1926-1932	445	3	0·67	545	18	3·3
1933-1934	566	8	1·41	528	8	1·52

It can be seen from the table that in the first series up to 1932 tuberculosis mortality is five times higher in the control than in the vaccinated group. But in the second series when the selection of infants for trial was improved, the mortality was the same in both the groups. Routine separation of the infants from their tuberculous parents before and after vaccination was not carried out with the result that some may have been infected before vaccination or shortly after the vaccination before the immunity developed. What the New York series suggest is that when B.C.G. is applied to a population where other measures of control maintain a low death rate and no precautions are taken to avoid infection before B.C.G. vaccine can become effective, the true specific effect of B.C.G. may be obscured.

(9) *B.C.G. in the Danish island of Bronholm*—Olsen (1943) reports that more than 23 per cent of the population has been vaccinated with B.C.G. and the whole situation on the tuberculosis front has changed. The number of recognised new cases of tuberculosis in the age groups between 15 and 35 has diminished considerably since the introduction of B.C.G. on a large scale. And it is in this age group that most cases of tuberculosis are found.

(10) *Trial on Nurses in Canada*—Ferguson (1946) carried this investigation on nurses in Saskatchewan hospitals and sanatoria. He has shown the diminution in tuberculosis morbidity with B.C.G. vaccine during the observation period in relation to the morbidity during the previous five years.

(11) *Vaccination of nurses in Stockholm*—Nordwall (1944) tried this in a training school for nurses at Stockholm. Before the introduction of B.C.G., 40 per cent of tuberculin negative nurses developed tuberculosis during service and had to break off from their studies for a more or less prolonged period, and one of them died. Amongst those vaccinated, there was only one case of pulmonary tuberculosis and she could resume service within a couple of months.

(12) *B.C.G. in other parts of the world*—In France, Weill-Halle quoted by Lotte (1946) states that among several thousand vaccinated persons over 25 years, he has never seen a case of clinical tuberculosis among vaccinated persons, even those living under conditions of exposure to infection, provided vaccination took place under conditions of freedom from contact and re-vaccination was carried out when indicated by loss of allergy.

In Great Britain, the application of B.C.G. has been officially approved. It has been extensively used in Switzerland, in Balkan States and in Poland. It has been recommenced in Vienna. Negre annd Bretey state that vaccination of new-born infants is now compulsory throughout Soviet Russia.

Various trials and investigations have thus been done and are being done in recent years with B.C.G. If the evidences that have already accumulated are carefully considered without prejudice one can conclude that B.C.G. is really efficacious in reducing the tuberculosis morbidity and mortality.

Difficulties and Disadvantages

(1) B.C.G. is a live vaccine and it has to be used within a fortnight of its preparation, though it should preferably be used in the first week. Care must be taken in its preparation to avoid contamination and in its distribution to make sure that most of the organisms are alive when the injection is made.

(2) The virulence of B.C.G. is not fixed and care must be observed that its virulence is not considerably altered. A nice balance is to be maintained between a degree of avirulence which renders it ineffective and a degree of virulence which leads to development of local lesions. This disadvantage, however, should not be over-emphasized. The Swedish B.C.G. vaccine was prepared from a strain which was obtained from Pasteur Institute of Paris in the year 1926, and the same degree of virulence has all along been maintained. Not a single case has been demon-

strated where the attenuated B.C.G. has regained its virulence.

(3) Local Lesions:—Local abscess and ulceration develop at the site of inoculation in a small number of cases and still more rarely there may be regional lymph gland enlargement and ulceration. The incidence of such local lesions varies widely with different workers and with the different methods of vaccination employed. If the inoculations are carefully made into the superficial layers of the skin, if the dose is not too high and if the organisms are not too virulent, the chance of abcess formation is little. When the abscess or the ulcer develops it usually heals up readily in the course of a few months, some of them requiring only scraping to facilitate healing. With multiple puncture method, the chance of local destructive lesion is practically nil, but it may produce up to forty rather angry looking papules which persist for weeks. This method, however, has the disadvantage that tuberculin sensitivity may not be encountered so constantly; and if Birkhaug's needle puncture apparatus is used there is difficulty in sterilizing it and in mass vaccination there is risk of virus hepatitis.

(4) Infants of tuberculous mothers are to be separated from their mothers at birth, until B.C.G. has made them tuberculin positive. If the infant or child is already exposed to infection, he is to be separated for at least six weeks more before vaccination to exclude the possibility of already acquiring the tuberculous infection. The separation of the infant from the tuberculous mother can, however, hardly be taken as a disadvantage exclusively confined to B.C.G. The infant in any case has to be separated from the infected mother, if the infection of the baby is to be avoided.

(5) Segregation of infants and children of tuberculous households presents a difficult problem to solve when the general vaccination of children is considered. Temporary removal of the patients to hospitals and sanatoria is difficult owing to shortage of beds. Segregation of infants and children in institutional accommodation is equally difficult. Further lodging of infants in large institutions involves the serious risk of cross-infection. The most satisfactory solution may be temporary removal of children or patients to the houses of relatives or friends.

(6) If the vaccination does not 'take', re-vaccination is to be done. It has been found that 2 to 5 per cent or more of all vaccinated require such revaccination. This depends to some extent on the method of vaccination. The general consensus of opinion is that the most consistent positive tuberculin reactions are obtained with intracutaneous method.

(7) Tuberculin sensitivity after vaccination is not of long duration. It may last for few to ten years or more. When the tuberculin sensitivity disappears, re-vaccination may be neecessary. If, however, the most susceptible age of childhood and adolesence is passed, re-vaccination is not so essential. In Sweden, such re-vaccination is not commonly done.

(8) Individual sensitivity to vaccine is variable. In a subject with low resistance, there may be abcess formation while in an individual with high resistance, there may not be any reaction whatsoever and probably it becomes ineffective in causing the specific immunity. Such variations are however rarely encountered.

(9) Vaccination of the new-born—Antigen developing mechanism of the new born is not well developed, so that often larger doses of B.C.G. are required to make them tuberculin sensitive and they take a longer time to become allergic. In Sweden, the practice is to vaccinate the new-born or very young infants with 0·1 or 0·2 mgm. of B.C.G. which produces about the same reaction as does 0·05 mgm in adults. Vaccination of the new-born has however the following advantages:

(*a*) Tuberculin-testing of the new born is not necessary before vaccination as all of them may be taken as tuberculin negative.

(*b*) Local abcess and ulceration occur very seldom in infants (Malmros).

(*c*) Babies born in the hospitals can easily be vaccinated there.

(*d*) Consent of the mother can be more readily obtained.

(*e*) After vaccination of the new born, subsequent tuberculin testing can be done without risk by Mantoux method direct with 1 mgm.

(10) When there is organization difficulty to vaccinate all the non-reactors, the vaccination may initially be confined to those with whom the risk of infection is particularly great. They are—

(*a*) New-born and non-reacting children living in tuberculous environments.

(*b*) Tuberculin negative nurses, medical students and other staff who should be vaccinated before they are allowed to join the hospitals and sanatoria.

Summary and Conclusions

A review has been made of the important investigations and trials that have been carried out in various countries in connection with B.C.G. The vaccine has got a definite and significant protective action against tuberculosis, though the duration and degree of immunity it confers is still unknown. Even though it does not give absolute protection, the tuberculosis developing after an adequately performed vaccination is of a very benign nature.

Its absolute harmlessness is proved. Not a single instance of progressive tuberculosis has been proved to be due to B.C.G. vaccine. In some cases in which the vaccinated developed tuberculosis, the bacilli have been found to be of human and not of bovine type.

Dangers and difficulties are there, but they can be overcome. With proper precautions, the chance of local abscess formation is little. The act of vaccination may be left with certain nurses or individuals who may be specially trained in this technique by practice with sterile saline. In general vaccination of children, the difficult problem to solve is segregation of infants and children from tuberculous households.

Mass vaccination is desirable. If that is not possible, the vaccination may initially be confined to the specially threatened section of the population.

Lastly, in a country like India where poverty, starvation, overcrowding etc., are there providing a soil for ready infection and its spread, where control measures of advanced Western countries cannot be introduced for years to come, a simple method of B.C.G. vaccination may prove to be of outstanding value as an economically practicable measure for effctive control of tuberculosis.

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CHRONIC DISCHARGING EAR

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In discussing the problems of discharging ear it is maintained that the discharge may come either from the middle ear or from the external ear. The middle ear discharge is more common, amongst the poorer section of any community. External ear discharge, or otitis externa is more common than otitis media amongst the Europeans in India. This phenomenon of racial discrimination of ear discharge came to the notice of the author during the last world war when he had the opportunity of treating ear, nose and throat diseases amongst Indian, British and African troops. Amongst the Indian troops otitis media was more common than the otitis externa, whereas amongst the British troops otitis externa was more common. This otitis externa had as well a seasonal incidence being more prevalent during the summer than during the winter months. And the troops used to call it 'summer ears'. Amongst the African troops, on the other hand it was almost unknown.

The external auditory meatus of the Europeans was found to be very narrow, so much so, that the smallest aural speculum could be put in the canal with difficulty. Whereas the external auditory canal of the African patients were so wide that membrane tympani could be visible, in some cases, without the help of a speculum. So it is maintained that wider the external canal less the incidence of inflammatory disease in that.

Regarding the seasonal incidence excessive perspiration in the summer, seabathing which the white troops used to indulge in, to cool their body surface, were the two contributory factors which would make the skin of the narrow external meatus soft and sodden. The superadded infection making it narrower still where products of inflammation could not come out easily. This would make a vicious circle and would perpetuate the thin watery discharge associated with aching pain in the ear with occasional exacerbation for months together.

Anatomy

A brief account of development and anatomy of the middle ear will help us in ascertaining the cause of continuance of the diseased process and assessing the best method of combating it.

The middle ear is formed with mesenchyme. The eustachian tube grows from the 1st branchial cleft. The blind end of the tube grows and passes through the mesenchyme and reaches the position of the aditus. During development the mesenchyme gets absorbed, and the eustachian tube dilates and fills the cavity thus formed. In infancy and childhood the blind end of the tube grows into mastoid antrum and mastoid air cells. The eustachian tube and middle ear cavity like the nasopharynx are lined with columnar ciliated epithelium. This ciliated epithelium is the only mechanism by which the secretion of the mucous glands or the product of inflammation can be removed from the middle ear tract. The ciliated epithelium and the mucous glands are more numerous at the pharyngeal opening of the tube than in the tube itself and the middle ear cavity. There are very few glands in the mastoid antrum and mastoid air cells. The cilia are absent in the mastoid air cells.

The tympanic membrane separates the middle ear from the external ear. It is 8-10 mm. in diameter. It has two parts, the membrana flaccida, above the two crescentic anterioposterior malleolar folds, and membrana tensa below the folds. The membrana flaccida has got mucous membrane inside and skin outside, whereas membrana tensa has got fibrous layer in between. The tympanum or middle ear is a six-sided box having the following dimensions: 2 mm. broad side to side opposite the umbo, 4 mm. at the lower part of the middle ear cavity and 6 mm. at the attic or epitympanum. Anteriorly, it is in communication with the nasopharynx through the eustachian tube. Posteriorly it communicates with the mastoid antrum through the aditus.

It will not be out of place to mention here that the eustachian tube is 36 mm. long in adults and makes an angle of 30° to the horizontal plane, whereas in children it is shorter, wider and less inclined. That is the reason why the eustachian type of otorrhoea is more common in children than in adults.

Etiology

It has been truly said that if one could treat the acute suppurative otitis media efficiently in time, the incidence of chronic suppurative otitis media would

have been very very less indeed. Therefore, the causes of chronic middle ear discharge are the same as those of acute middle ear suppurations. The following are some of the factors to be considered in this connection:

(*a*) Lack of drainage of the products of inflammation from the middle ear cavity.

(*b*) Scarlet fever, measles and diphtheria are usually followed by middle ear discharge.

(*c*) Resistance of the patient: If the resistance is lowered by long continued disease, *e.g.*, diabetes, tuberculosis, etc., middle ear discharge does not dry up if the general health is not improved.

(*d*) Poorer section of the population suffer more than the wealthy, because the latter would not allow acute inflammation to become chronic.

Signs and Symptoms

The following signs and symptoms are met with in middle ear suppurations: discharge, deafness, pain, tinnitus, vertigo and perforation of the membrana tympani.

(a) *Discharge*—(*i*) Thin watery discharge is usually met with in early cases of tympanic type of otorrhoea—with efficient treatment the discharge dries up leaving a small scar on the membrane with no impairment of hearing.

(*ii*) Thick, mucoid or mucopurulent discharge in abundance found in eustachian type of otorrhoea.

(*iii*) Thick purulent discharge, foul-smelling—

(*a*) Small in quantity—mastoid type.

(*b*) Large in quantity with or without blood stain—polypoid type or all-involved type with or without polypus or granulations.

(*iv*) Thin purulent discharge with scales of desquamated epithelial debris—very bad-smelling—attic type of otorrhoea.

(b) *Pain*—It is not a common symptom of uncomplicated chronic otorrhoea. Simplest cause of pain may be retention of the products of inflammation, through the perforation which is small, or it may be a precursor of intracranial complication. So every case of chronic middle ear discharge associated with pain and tenderness over the mastoid, should be thoroughly investigated by the expert.

(c) *Deafness*—It is not a marked symptom in early cases of chronic otorrhoea. In late cases of 3-4 years' duration deafness may be marked due to fibrosis of the stapedio-vestibular joint.

(d) *Tinnitus* or noises in the ear—It may or may not be present in chronic otorrhoea cases.

(e) *Vertigo*—It may be due to retained secretions pressing on the oval and round window of the internal ear or it may be due to erosion of the bone of the internal ear exposing the endosteum to atmospheric pressure. The latter case is a serious complication of chronic middle ear discharge and known as fistula syndrome.

(f) *Perforation*—It may occupy different position of the tympanic membrane. Proper location of the perforation will help us in the diagnosis, prognosis and treatment. All these different types of perforation can only be recognised and a proper diagnosis of the type of otorrhoea made with the help of an auroscope. Without proper diagnosis one is liable to substitute the treatment of one type with that of the other type and one is sure to waste valuable time and incur displeasure and annoyance of the patients and their relations. In some cases there may develop some intra- or extra-cranial complications.

With an electric auroscope or a properly reflected light one should proceed to examine the case after clearing the external meatus by mopping with sterile cotton wool and pulling the pinna upwards and backwards and introducing the largest size speculum which the ear would accommodate. One of the following types of otorrhoea may be found in the tympanic membrane:

Varieties

A. Tympanic Type.

(1) Central kidney-shaped or round perforation. There is no polypus or granulation, no tenderness on the mastoid. The nose and pharynx are fairly healthy. This is tympanic type of otorrhoea. Here the sepsis is confined to the tympanum only. If one can eradicate that sepsis the ear will be dry and the perforation will be healed by scarring with no impairment of hearing, provided the perforation is small, *i.e.*, size of a pin's head or a bit larger.

If it is larger it will not heal by scarring but the aim of treatment in these cases would be to make the ear dry and keep it dry.

There are different methods of treatment:—

(*a*) Wet treatment—(*i*) Syringing the ear with sterile normal saline or 2·5 per cent boric acid solution, (*ii*) Using H_2O_2 drops in the ears.

Contra-indications: (1) Febrile cases. These cases suggest that pus is pent up and so it has got to be evacuated by operations of myringotomy, mastoid operation or exploration of the lateral sinus. This will tide over the need of operation but may frequently give rise to intracranial complications.

(*ii*) Acute otitis media or acute exacerbation of chronic suppuration. Secretion in these cases has got high bactericidal power. We should not remove it by dilution.

(*iii*) Incipient mastoiditis or any sign of intracranial complication. It is better to operate on these cases and get the patients free from potential danger than to treat them by palliative measures. The syringing fluid and H_2O_2, as a rule cannot enter the mastoid antrum by ordinary syringing and cannot cause resolution of the inflammation. If on the other hand it (syringing fluid and peroxide) penetrate the antrum it may as well pass beneath carious bone and spread the infection to the areas which have been healthy uptill now.

So syringing the ear or putting in of H_2O_2 in the ear is never encouraged. Cleansing can be effected by mopping with sterile dry cotton. In majority of complicated cases this mechanical removal of pus will be sufficient to effect a cure of middle ear discharge. In some other cases instillation of drugs into the ear will be helpful. The following prescriptions may be of use in uncomplicated cases.

(1) Liquid paraffin prevents dermatitis of the external ear.

(2) R/ Menthol—10 grains
Liquid paraffin—1 oz.
Antiseptic.

(3) Alcohol is a powerful antiseptic:
R/
Spt. Vini rectificatus—½ oz.
Glycerine—1 drachm
Aqua—1 oz.
Early stage.
or
Spt. vini rectificatus—late stages: causes pain; as in the case of recovery it is stopped altogether.

(4) Carbolic acid—Analgesic
R/
Glycerin acid carbolic—2 drachms.
Glycerine—1 drachm.
Aqua—1 oz.

(5) Sodi bicarb—To dissolve thick inspissated pus
R/
Sodi bicarb—30 grains.
Glycerine—2 drachms.
Aqua ad.—1 oz.

(6) Acid treatment—when pus is highly alkaline.
R/
Acid boric—10 grains.
Glycerine—1 drachm.
Aqua—1 oz.

Glycerine acid boric is a mild antiseptic but its beneficial effect is due to its acidity. Boric acid can also be insufflated into the ear. It helps in soaking pus.

(7) Calamine—In cases where *external otitis* is also present.
R/
Calamine—½ drachm.
Glycerine—½ oz.
Water—1 oz.
or
R/
Calamine—½ oz.
Liquid paraffin—1 oz.

There are other favourite prescriptions, *viz.*—(1) 1 per cent solution of perchloride of mercury, (2) 1 per cent solution of picric acid in 70 per cent alcohol, (3) 2½—5 per cent of lotio mercurochrome in aqua distillata. These prescriptions are of secondary importance in treating the otorrhoea of this type (tympanic). The main point is mopping and draining it as often as necessary during the day to be followed by any mild antiseptic ear drops.

Penicillin—Instillation of penicillin into the ear and injection of the same have not been found to be useful in the treatment of chronic middle ear suppuration in my practice.

(*b*) Dry method. Here the ear is mopped dry by the sterile cotton wool and some powdered medicine is then insufflated inside the middle ear cavity under vision. The following powder may be used.

(1) 75 per cent iodine resublimate in boric acid ampoules freshly prepared powder should be used, if we want to have the effect of iodine. Otherwise iodine will evaporate leaving boric acid.

(2) Acid boric—powder. They are mild antiseptic and absorbs the secretion.

(3) Any sulpha preparation:—
(*a*) Cibazol antiseptic powder.
(*b*) Sulphadiazine with flavin etc.
We need insufflator for this purpose.

Electric method. This is highly technical requiring special appliances and skill. The main principle is to dissociate the Zn ion from the sulphate by passing electric current through the zinc sulphate solution put into ear. Ionisation forms an antiseptic barrier between the tissue and exterior and gives the tissue rest and time to heal up.

The methods of treatment mentioned above will result in cure of 50 per cent of all cases of otorrhoea. 25 per cent will require special kind of treatment, *viz.*, electric ionisation etc. Another 25 per cent will require cortical mastoid, modified radical or radical mastoid operation.

Prognosis:—
Hearing—good.
Life—good. No danger of intracranial complication.

B. EUSTACHIAN TYPE.

This type is met with in childhood. The discharge is whitish, thick, ropy and mucoid and never bad-smelling. On examination we find an anterior and inferior perforation of the membrane. The child is generally a chronic sufferer of common cold and cough. The usual history in these cases is that the running from the ear is preceded by an attack of sore throat or common cold which subsides after a few days but the running continues for sometime. If the cases are examined at the early part of the attack we find a small perforation in the anteroinferior quadrant of the membrane with pulsating discharge indicating acuteness. During quiescent period the ear may show a thin scar on a small patent perforation. In children invariably we find signs of chronic tonsillitis and adenoids with enlarged neck glands. Adults who are not so common victims of this type of otorrhoea may show signs and symptoms of sinusitis.

Treatment surely consists of dealing with the source of sepsis. Tonsillectomy and adenoid curettage followed by ordinary treatment by mild antiseptic ear-drops etc., will improve the ear condition and give a dry ear. The following note of warning is worth mentioning:

If the case has been neglected for long, giving a big perforation in the ear we may not get a permanent dry ear. The patient should not allow water to get into the ear, which will start discharging as soon as water comes in contact with the middle ear. So the following precautions must be taken by the patient.

(1) Not to go for bathing in tank and river or seas etc.

(2) Protect the ear always with a little pledget of cotton wool to act as filter of dirt and dust.

(3) Soak cotton wool pledget in oil while taking bath.

After the ear has been kept dry for a couple of weeks and if we find the perforation is small in size, we may try closure of the perforation by inducing fibrosis. This can conveniently be done by touching

the margins of the perforation with some caustic *i.e.*, trichloracetic acid, chromic acid etc. But this should always be left for the experts.

Prognosis:—
Hearing—good if treatment is undertaken early.
Life—good *i.e.*, complications are rare.

C. Mastoid Type.

The perforation is in the posteriosuperior quadrant of the membrane. The discharge is thick, purulent, may be scanty or abundant, and bad-smelling. Hearing is not affected if the abundance of discharge does not mechanically cause obstructive type of deafness.

If the discharge is in good quantity we invariably find a little granulation tissue or polypus in the neighbourhood of the perforation. Even if nothing is visible to the naked eye, except a marginal perforation, x-ray would show an underlying caries or necrosis of the bone, or even mastoid may be affected.

The treatment in this type of cases is operative. Granulation or polypus should be removed by operation and if x-ray does not show any impairment of the mastoid air cells, treatment to be continued on the conservative line as detailed before. If mastoid cells show signs of necrosis the diseased bone must be removed by cortical or modified radical mastoid operation.

D. Attic Type.

In these cases perforation is present in the membrana flaccida. The discharge is usually scanty but smells very badly. The patient suffers from occasional attacks of giddiness. In a case it was reported that pressure on the tragus while shaving caused giddiness and the patient lost his balance and fell down on the floor. On examination an attic perforation was found. Increase of intratympanic pressure caused giddiness. These cases are usually associated with cholesteatomata. A thick cheesy material is found at the attic region. It is never found in central perforation. It is formed by the desquamation of the epithelium of the skin of the drum, which grows inside through the margin of the perforation. These scales are arranged concentrically and here and there crystals of cholesterin may be found. They increase in size by successive accumulation and erode the bone of the semicircular canal by pressure, exposing the endosteum, to the external air pressure which when raised and conducted to the perilymph causes giddiness.

Treatment:—Conservative treatment here is very unsatisfactory. Some suggest intratympanic syringing of the parts through attic canula, others advise injection of the carbon tetrachloride—but it is of no use. Operation is the only treatment that will be of avail.

Modified radical mastoid operation aims at eradicating the disease and saving the patient from potential intracranial complication.

Prognosis:—Unceratin The patient is liable to develop intracranial complication, cerebral abscess, meningitis etc., at any time.

E. Polyroid Type.

All the structures of the middle ear cavity are involved here—ossicle, mucous membrane of the tympanum, tympanic membrane etc. The ossicles may be necrosed or a polypus is developed from the inner tympanic wall protruding into external ear through the perforation. The patient usually gives a history of discharging ear for a pretty long time. The discharge is usually foetid, large in quantity and blood stained. There may or may not be tenderness over the mastoid border or tip depending on the thickness of the outer table of the mastoid. These cases are almost always associated with chronic mastoiditis. Diagnosis must be clinched by skiagraphy.

Prognosis:—Uncertain, unless treatment is undertaken early, patient would lose his hearing, and his life may be endangered by intracranial complications.

Treatment: Conservative treatment is of no avail. Operation: Probably radical mastoid operation, would retain the residual hearing and save the patient from potential intracranial complications.

Specific infections of the ear having chronic discharge are: (1) Diphtheria; (2) Tuberculosis; (3) Syphilis; (4) Scarlet fever.

(1) Diphtheria: The discharge is intractable, whitish in colour and highly alkaline to litmus. The infection is secondary to faucial or nasal diphtheria. Faucial diphtheria is apt to disappear and its absence is the cause of difficulty in diagnosis. Nasal diphtheria, gives whitish mucoid discharge from the membrane present at the posterior part of the nasal cavity. Treatment—(*i*) Glycerine and boric acid drops in 1 per cent citric acid; (*ii*) Ionisation.

They do not cause diphtheria to other persons who come in contact as they are less virulent to cause infection.

(2) Tuberculosis: Two kinds:—
(*a*) Pyogenic infection on tuberculous soil.

Membrane is pallid and shows very little reaction to infection.

Treatment: Improve the general condition of the patient by sanatorium type of treatment. If mastoid is affected—operation of mastoidectomy—but the healing is very slow.

(*b*) Infection with the tubercle bacillus—(*i*) Through eustachian tube (*ii*) through blood stream.

Painless central perforation with scanty mucoid discharge; membrana tympani is pallid, perforation advances with great rapidity—ultimately the whole of the membrane is removed. Multiple perforation in

the M.T. is not pathognomonic of tuberculosis. Inner tympanic wall is pallid. There may be sudden unheralded tuberculous meningitis.

Treatment where active focus is found anywhere in the body. Sanatorium type. The resistance of the patient is raised. If the ear becomes quiescent for a couple of years it will not recur.

If the ear is the primary source of infection it will have to be operated on to remove bone infection etc. The bone is found to be in the stage of extreme destruction and ischaemic. It has to be removed until we reach an area which blinds.

(3) Syphilis. All the signs and symptoms of pyogenic infection associated with intractable nerve deafness—give the clue to the diagnosis. Treatment: Antisyphilitic and local.

(4) Scarlet fever. It is uncommon in our part of the world. Suppuration may accompany or follow the disease. Treatment is just like that of pyogenic infection. If not successful, at an early date mastoid operation of cortical type should be undertaken.

Otitis Externa

(1) Diffuse otitis externa: It is generally secondary to chronic otitis media, may also be associated with spirochaete and fusiform bacillus. Treatment: Application of equal parts of liquor arsenicalis, vinum ipecac and glycerine.

(2) Dermatitis or seborrhoeic eczema: It is generally found in association with seborrhoeic dermatitis of the scalp and auricle. The canal is filled with epithelial debris. There may be narrowing of the meatus.

Treatment consists in cleansing the ear canal by syringing to be followed by dehydration with alcohol and drying. The application of the following may also be tried in intractable cases, *e.g.*

(*a*) Silver nitrate 15 grains, Spt etheris nitrosi 1 drachm.

(*b*) Salicylic acid 10 grains, sulphur ppt. 10 grains, vaseline 1 drachm., on a small piece of gauge.

(*c*) 5 per cent sulphathiazole in vaseline in gauge etc.

Otomycosis: Presence of tinea, Aspergillus Niger in the external auditory meatus, causes deafness and extreme iching sensation, so much so, that the patient says that he would like to tear the inside of the ear out to get relief.

On examination: Brownish black mass is found in the deeper part of the meatus. If it continues for some time the tympanic membrane may also be permanently damaged.

Treatment lies in syringing out the epithelial debris and drying it with alcohol and touching the parts with triple dye, *viz.*, methylene blue 1 per cent., gentian violet 1 per cent., and brilliant green 1 per cent in spirit rectificatus.

Sometimes 2-3 syringings and applications may be required.

CONVULSION THERAPY IN PSYCHIATRIC CASES*

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Convulsions are produced in two ways—*viz.*, (1) Chemically and (2) Electrically.

Chemical Convulsion

History—Von Meduna, a psychiatrist of Budapest observed that schizophrenics rarely suffer from epilepsy and two schizophrenics who developed convulsions during the course of their disease definitely improved of their mental conditions. Thus he concluded that there might be a biological antagonism between schizophrenia and epilepsy, though epilepsy in the heredity is in many occasions precursor of mental diseases. This idea led him to think about the artificial production of epileptiform convulsion in schizophrenic patients and he, in 1934, introduced cardiazol convulsion therapy with marked benefit. Cardiazol stimulates the vasomotor centres and produces vasoconstriction of the blood-vessels of the brain and thereby gives rise to convulsions.

Technique—The method consists in the intravenous injection of a ten per cent solution of Cardiazol (Leptazol B.P.)—chemical name pentamethylene tetrazol. The injection is given two or three times a week usually in the morning on a fasting stomach. The patient is placed on padded wooden table with a hard pillow under the dorsal spine to prevent vertebral fractures. Metal articles, such as artificial dentures, hair pins are removed, belts, strings and straps are made loose. A mouth gag is given to prevent the biting of the tongue as soon as the patent opens his mouth. The commencing dose is 0·5 gm., *i.e.*, 5 c.c. of a 10 per cent solution in case of male patients and 4 cc in case of female patients. The injection is given rapidly in a wide bored needle.

The fits—There are three stages to the fits produced:

1. Pre-convulsive stage,
2. Convulsive stage,
3. Post-convulsive or recovery stage.

1. Pre-convulsive stage. In about two to five seconds the face of the patient becomes flushed and sometimes he coughs. Next two or three seconds the patient experiences some peculiar discomorts of the types of respiratory choking, and also experiences an unknown fear of death. Then suddenly he becomes unconscious and gets a violent jerk from the sudden tonic spasm of the whole body with marked rigidity. The mouth is opened, the legs drawn up, the wrists are flexed.

*Read at the Science Congress, Patna, 1948.

2. Convulsive stage. The tonic contraction is broken up by the clonic jerks after about fifteen seconds, and these continue for a further thirty seconds, when respiration completely stops and the whole body is cyanosed. Patients in this stage occasionally lose control over the sphincters and may get discharge of urine, fæces and semen. Clinically pupils are dilated and not reacting to light. The corneal reflex is lost, other reflexes are brisk and the plantar flexor. There is rise in pulse rate and increase in blood pressure.

3. Post-convulsive stage. Recovery from convulsion varies in different patients, but is characteristic of each. Some sleep for several hours, others are delirious and restless. Some are confused, not knowing where he is and what he is doing and are usually found to be abusive and assaulting. Some become erotic, others apologetic or weeping. Some walk away straight from the table but others get retching and vomiting. Usually in about ten to fifteen minutes the patient becomes normal and fit to be left alone.

Physiological changes during the fit—Besides rise in pulse rate and B.P. the following changes occur—

1. Blood sugar either rises or falls in pre-convulsive stage—reverse in convulsive stage.

2. Potassium level falls.

3. Calcium concentration rises.

4. C.S.F. barrier to bromide ions is lowered.

Careful physical examination should be made before proceeding to convulsion shock therapy. This treatment should be conducted with the greatest consideration by those who have carefully witnessed convulsion therapy in several cases. and should be made only in carefully selected cases.

Complications of Treatment—1. Loss of memory —During the time of convulsion and sometimes the memory of minor events near about the time of convulsion is also impaired.

2. Ligamental tears and dislocation of the mandible, shoulder and hip.

3. Vertebral fractures—varies from slight crush of the body of the vertebræ to a telsecoping of vertebral body.

4. Occasionally fracture of the long bones.

5. Cerebral haemorrhage.

6. Rarely lung abscess and embolism have been recorded.

7. Danger of flare up of latent tubercular infection.

Electrical Convulsion

History—Hitzig in 1870, was the first to use electricity as a means of stimulation of the exposed cortex in animals. Later W. Grey Walters (Bristol) points out that electrical stimulation of the brain may produce epileptic fit but as the brain is efficiently protected by the poorly conducting scalp, skull and meninges, a strong current must be applied to convey the adequate stimulus to the brain. This would be painful to the skin and might end in fatality, hence the idea was left for a long time until in 1938 L. Bini and V. Cerletti of the University of Rome first devised an apparatus for clinical application of electric shock to schizophrenic patients without any accident. They claimed this porcess of induction of epileptiform fits is much safer than the Cardiazol method and at the same time yielding the same amount of benefit.

Technique—All the instruments henceforth devised are modelled after the original Bini-Cerletti type by which the alternating current delivering between 80 and 200 volts and 200-700 ma (milliampere) is applied for 0·1 to 0·5 seconds. The dosage may be varied. The patient is placed like that in chemical method on a firm bed. His auriculo-temporal and suprazygomatic regions are cleansed with soap water and spirit lotion. Then the electrodes covered with thin linen and moistened in 20 per cent warm saline solution are applied over the two temporal regions with firm pressure. Transtemporal resistance of the head is measured by the instrument. Then the instrument is adjusted with the same resistance. The switch is changed from the test circuit to the treatment circuit and the automatic stop switch set at 0·1 second. A rubber gag is placed between the patient's teeth to prevent the biting of the tongue and breaking of the teeth and also to keep the air passage open.

Complications are similar to those of chemical shock treatment but are less frequent. The advocates of the electric shock therapy claim that death occurs only rarely.

The advantage of the electric shock therapy over the other convulsive methods are—

1. The patient instantaneously becomes unconscious, so does not experience pain or fear of death and does not therefore resent treatment.

2. Since no toxins are introduced into the system pulmonary and myocardial complications are avoided.

3. Psychomotor excitement similar to that following cardiazol convulsion is of less intensity.

4. Difficulties involved in intravenous medication do not arise.

5. If no convulsion ensues, treatment can be immediately repeated without any resentment of the patient.

6. The method requires little time and expense.

Clinical Uses

From experience it is seen that convulsion therapy acts particularly well in catatonic types of schizophrenia. Here a case may be described. A rating in a merchant navy was waiting for a long time in a military hospital for disposal difficulty. This man was given four convulsive shocks after which he behaved perfectly normally for about two and half months.

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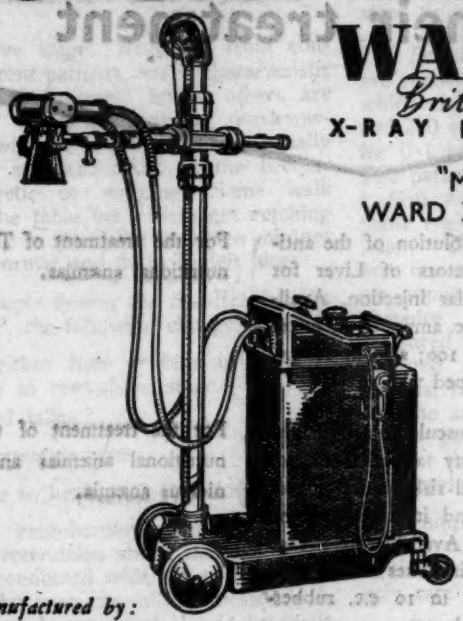

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Then he ceased to talk to others and refused to take food but for two or three days continued to go to the garden with other patients to attend the routine occupational work. Some food could be fed by coaxing for a day or two but later he absolutely refused to take any food, did not get up from his bed, nor opened his eyes, lay always on his own cot without any sort of movement or response to questions. He had to be fed with nasal tube. He was given convulsion shock with cardiazol. The same evening he got up from bed, opened his eyes and came out on the verandah and sat down there quietly, responded to questions very slowly in a low tone. Next day he could be coaxed to take his food. On the third day another shock was given, the same evening he was found to follow others—to go and bring tea and evening meals. Since then he was behaving quite normally but did not like to have further shocks. No shock was given for a couple of months until he relapsed into the catatonic stage again. This time also he responded well with one shock. In this way I had the opportunity to observe the case for about ten months during which time he had several attacks but every time he responded well with one shock.

In other varieties of schizophrenics, definite improvements are noticed in early cases, although exceptions have been noted.

Several melancholias and both phases of maniac depressive psychosis respond well.

A robust Sikh patient belonging to the Indian Pioneer Corps was very quarrelsome, and abusive and off and on used to become violent and assault other patients. He was forcibly given a course of four shocks after which he became quiet, ceased to quarrel and fight with others and became co-operative and useful to Ward works.

Convulsion therapy has also been tried in psychoneurotic cases, especially in those obsessional cases who are hot satisfied with the general psychological treatment, but repeatedly demand for some drastic measures like abdominal operation, and also in those cases who firmly believe that they are not properly cared for or treated efficiently. Convulsion in many of these cases produce marked benefit. Action is probably due to suggestion.

A patient repatriated from overseas had some abdominal pain. The case was thoroughly investigated by the medical and surgical specialists both at overseas and at the base hospital in India. Nothing organic was found responsible for his trouble. Finally he was referred to the psychiatric department for necessary treatment and disposal. He had been diagnosed to be a case of Hysteria, but when general psychological treatment failed, convulsion therapy was attempted on him. But to our surprise he felt eighty per cent relief after the first shock and after the second shock three days later he completely recovered. He was co-operative and very keen to serve the Army. He was recommended leave and downgraded to category C, *i.e.*, service only in India.

SPECIAL ARTICLE

OBSERVATIONS ON CHOPRA COMMITTEE'S REPORT

A MEDICO

Analysis of the Report and Recommendations

The report of the Committee on the Indigenous Systems of Medicine comprises two volumes; the first volume contains the report proper and the Committee's recommendations, while the second volume consists of appendices which deal mostly with the views expressed by different witnesses whom the Committee examined.

The subjects dealt with in the first volume are:

1. The historical aspect of the indigenous systems of medicine.
2. Consideration of the reports of previous Committees appointed by various Governments to report on indigenous systems of medicine.
3. Existing conditions of medical relief in India.
4. Methods to be adopted in the synthesis of Indian and western medicine.
5. Training particularly with respect to synthesised medicine.
6. Organization of rural medical relief.
7. Need for State control of medical practice and education.
8. Research.
9. Financial considerations.

Terms of Reference—According to the original terms of reference the Committee was asked to make recommendations with respect to the provision of research in, and the application of, scientific methods for the investigation of indigenous systems of medicine and to put up proposals for improving the facilities for training in Indian systems of medicine and to make provision for State control of practice in these systems, through registration. The Committee apparently seems to have extended the terms of reference to include the suggestion made by the Hon'ble Gazanfar Ali Khan, the then Minister for Health, to explore the possibility of combining all the systems of medicine into one all-comprehensive system. The Committee has actually expended most of its efforts in finding out a suitable formula for the integration of the different systems of medicine. The report, therefore, from the beginning to end is calculated to prove and establish the desirability and the practicability of bringing about a synthesis of different systems.

Present state of Indian Medicine—In their chapter on the existing conditions of medical relief they speak about the present state of the indigenous systems of medicine in India and show how *hakims* and *vaids*, who mostly live in the villages, and who are more "sympathetic and personal" and who deal in "locally available and inexpensive drugs, and preparations", and above all who use "the local language", are better fitted to treat the Indian villager, whose constitution, according to the Committee, is more suited for being treated with drugs of the Indian system than those of the modern system. The Committee estimates that there are between 200-250 thousand *vaids* and *hakims*

in India. Of these only 51,700 are reported as registered. There are 51 hospitals and 3,890 dispensaries under the indigenous system catering roughly to over 15,000,000 people annually. There are about 57 educational institutions giving training to 3,133 students yearly.

Integration—On the most important question of synthesis of Indian and western medicine the Committee has made the following recommendations:—

NEED AND PRACTICABILITY. They believe that, while Indian medicine can take much of practical value from western medicine, the latter can also learn much from the philosophic background of Indian system, its comprehensiveness, the importance it attaches to the soul factor and dietary, its generalisation of principles and the knowledge of truth gained by the use of "supersensory perception." The Committee emphasises a great deal upon the aspect of intuition which, according to them, is of much greater value than all the modern methods of diagnosis, which depends a great deal upon laboratory investigations and radiological examination.

2. IST STEP. The first step to be taken towards integration is to teach subjects pertaining to both modern and Indian systems of medicine side by side in all teaching institutions. At this stage, according to them, the process will be one of supplementing and not of real integration. Hence during the college courses 10 subjects beginning with basic sciences and ending with medical jurisprudence will be taught according to both Ayurvedic as well as modern systems.

3. 2ND STEP. The second step will be the teaching of each subject by the same teacher instead of by two or more as previously, which will give the students a unified view of the Indian and western medicine.

4. 3RD STEP. The final step will result from the work in the research institute where eminent experts of western and Indian systems will work side by side discussing, testing, checking and verifying the various hypotheses and theories with a view to either rejecting or harmonising them.

5. In order to facilitate the integration of teaching and studies the Committee recognise that further steps should be taken simultaneously, namely, improvement in the basic qualifications of students, compilation of special text books for integrated studies and training of "synthetic" teachers.

Medical Education—As regards training the Committee has suggested that 31 colleges of Indian medicine that are at present functioning in different parts of India should be upgraded in order to enable them to admit 75 students each year. In that way they expect to have 1,350 graduates turned out annually from these colleges. Each of these colleges should have 32 teachers for teaching subjects in modern medicine and 32 teachers for teaching subjects in Indian medicine. The Committee also suggests that for qualified practitioners of modern medicine special courses of instruction in Indian medicine should be instituted. This should extend over a period of two academic years. They also recommend a course of study extending over a period of two academic years for practitioners of Indian medicine in subjects pertaining to modern medicine.

Rural Medical Relief.—In the chapter on organisation of rural medical relief they have formulated what, in their opinion, is a comprehensive plan which could be put into effect economically and expeditiously. In order to produce the necessary medical personnel the Committee suggests that a three-year diploma course should be introduced and existing practitioners of Indian medicine should be utilised by giving them necessary training in public health, surgery and obstetrics for a period of six months. They estimate that in five years' time at least 25,000 of these hakims and vaids could be given this short term training and utilised for manning rural dispensaries. The Committee also suggests that they should be given a salary of Rs. 75-150 per month. The complete scheme of health organisation consists of:

	Total number for the whole of India.
1. Rural dispensary or primary unit 1 for 3,000-4,000 population	100,000
2. Secondary units—1 for every 10,000	30,000
3. Panchayat dispensary with a mobile unit—1 for every 50,000	6,000
4. Tahsil hospital with 30 beds for every 100,000	3,000
5. District hospital with 250 beds 1 for every 10,00,000 ..	300
6. Provincial Hospital with 2,000 beds.	

State Control—On the question of State control the Committee has suggested that adequate provision for the medical education of all the recognised systems should be made, that all practitioners should be registered on an all-India basis, that there should be disciplinary control over practice and that consultative and advisory bodies should be constituted for giving advice in matters pertaining to public health and medical relief. The Committee suggests that at present there should be two registers, one for practitioners of modern medicine and the other for those practising Indian medicine. In their opinion both qualified and unqualified *vaids* and *hakims* should be entered in a single register. There should be, however, separate electorates for representation in the Councils.

Research—As regards research the Committee is of opinion that it should have the two-fold aim of clearing Indian medicine of accretions of centuries of doubtful value and to work for a synthesis of the Indian and western medicine so as to evolve a unified system of medical relief and education. With these objects in view the Committee suggests that research should be conducted under the following categories:

1. research in the fundamental doctrines of Indian medicine;

2. literary research;
3. clinical research;
4. medical research;
5. research in nutrition and dietetics;
6. research of psychological aspects of medicine.

They recommend the establishment of a Central Research Institute with clinical, laboratory, pharmaceutical, library, statistical and museum sections.

Finance—The Committee suggests that each of the existing educational institutions should be given a capital grant of Rs. 2½ lakhs and a recurring grant of Rs. 1½ lakhs in order to enable them to be upgraded. Apparently they suggest only 10 institutions to be upgraded as they have calculated the total expenditure involved as Rs. 25 lakhs non-recurring and Rs. 15 lakhs recurring. They also recommend a subvention of Rs. 30 p.m. for each trainee for the rural medical scheme which would cost Rs. 1,20,000 for each provincial government for training 600 practitioners every year. The expenditure in regard to the Research Institute is estimated at Rs. 5 lakhs non-recurring and Rs. 2½ lakhs recurring.

The Committee also recommends that there should be a Deputy Director General of Health Services appointed under the Ministry of Health to hold charge of the section on Indian medicine.

Objections to the Report

Term of Reference overstepped—A technical objection can be raised against the report of the Chopra Committee in that they have gone beyond the terms of reference by suggesting proposals for integration of indigenous and modern systems of medicine. According to the original terms of reference the Committee has been asked to report mainly on measures to be taken to improve the Indian systems of medicine by conducting research on scientific lines, by raising the standards of education in those systems, and by introducing measures for exercising State control over the profession which practises the indigenous systems. Instead of confining themselves to the main terms of reference the whole report is a plea from the beginning to end to bring about a synthesis between the indigenous and modern systems of medicine.

Synthesis by stages unworkable—The Committee has suggested that synthesis should be brought about in three stages. In the first stage Indian and modern systems of medicine should be taught side by side in educational institutions by separate groups of teachers. In the second stage both the systems of medicine should be taught side by side but by the same group of teachers. The final stage is the process of research by teachers in both the systems of medicine in order to clear Indian medicine of 'accretions of centuries' and to find out and establish the truth or otherwise of the existing theories on which Indian systems of medicine are based. In more than one place have the Committee accepted the fact that Indian systems of medicine have been in a state of stagnation for centuries and are today covered over by an accumulated mass of accretions. In the circumstances it is not understood how and why the Committee should suggest that the first process in bringing about a synthesis of indigenous and modern systems of medicine should be the unjustified burdening of the curriculum of studies for the medical student with exploded theories of doubtful value. On the other hand the more rational approach towards synthesis would be first to conduct research and subject all the theories on which the indigenous systems of medicine are based to careful scrutiny and investigation on modern scientific lines and then incorporate whatever is found to be of value, in modern medicine. No one will deny the fact that modern medicine is but a stage of evolution of the systems of medicine which were practised in ancient times. Ayurveda can justly and proudly claim to be the foundation of all systems of medicine. The Committee accepts the fact that there cannot be two sciences. Science is one and indivisible. So long as medicine is a science there can be only one medical science. The principles and practices of Indian medicine were in the days of old accepted by other countries who by force of circumstances have been placed during recent years, in a more favourable position for the development of science as a whole. Medical science had therefore ample opportunities in western countries to develop into what it is today. No doubt the development of modern medicine in those countries was based originally, centuries back, upon the system of medicine that had perhaps its first roots in India. Unfortunately medical science which had its beginning here was not given the opportunities for development in this country. Hence it remained stultified and stagnated. Centuries ago when Indian medicine spread to other countries in the west, all the principles and theories of Ayurveda, namely, Darsana, Panchabhootha and Thridosha theories were all accepted as gospel truths. They, however, did not stand the test of time when basic sciences developed in western countries and their principles were applied to establish the truth or otherwise of the various theories upon which the Indian system of medicine was originally based. I do not, however, want to accept the verdict of western nations on the principles and theories of Indian medicine. I would however urge primary need for clearing the ground ourselves by conducting research on modern scientific lines and finding out the value of these theories. Until that is done it will be criminal waste of time and energy to overburden the curriculum of medical studies with parallel courses in both systems of medicine. If the real aim of the Committee is to bring about a rational synthesis by which is meant taking in all that is good in both the systems of medicine and discarding what is bad, they will be defeating their purpose, if the process of integration by the stages they have suggested is put into practice. If it is an accepted fact that the indigenous systems of medicine are covered over by accretions I cannot possibly understand why the Committee recommends that acquirement of knowledge of such useless accretions should form part of the training programme for doctors.

Science has no geographical boundaries. It is universal. It should not be governed by narrow nationalism. The Indian system of Ayurveda in ancient times was gladly accepted and adopted by other countries and not rejected on the plea that they were not indigenous to those countries. If modern medicine, which has been allowed to grow and develop in western countries owing to the opportunities they had for the development of basic sciences as a whole, there is no earthly reason why we should not adopt it and call it our own for the simple reason that modern medicine has itself sprung up from ancient Ayurveda, which had its beginnings in this country. It is quite possible, however, that some of the theories as well as some of the very useful medicines in Ayurvedic pharmacopoeia will stand the test of scientific scrutiny. Hence the first step in the process of integration, if there is any need for it at all, should be research on indigenous systems of medicine. For this purpose we can certainly establish either central or provincial research institutes whereby the first term of reference to the Chopra Committee can be given effect to.

I am against the process of integration as suggested by the Chopra Committee, though I am in full agreement with their recommendations regarding research. The question, however, will naturally be asked as to what should be done with the large body of practitioners of indigenous systems of medicine and with the institutions, both private and State, which have been established for the purpose of giving training in these systems. I consider that it should be but fair to allow these practitioners to practise and the institutions to impart instruction in Indian systems, until such time that research conducted on indigenous systems reveals the efficacy of some of its principles and theories. If research has been able to establish that the basis of indigenous systems of medicine is sound, we should not hesitate to give all help possible for their development. If, on the other hand, only a few theories and some of the medicines that are used in indigenous systems of medicine have been found to stand the test of scrutiny, I suggest that the systems should cease to exist as such, but whatever has been found valuable should be integrated with modern medicine. In such a case all the institutions which give training in the indigenous systems of medicine should be closed and a short period of 10 years should be given for the practitioners of Indian systems of medicine either to learn the practice of modern medicine or cease to practise the indigenous systems at the expiry of that period. We have the examples of Japan and China for adopting such a procedure. Chinese medicine is perhaps more ancient than Indian systems of medicine. Chinese medicine as it was practised in ancient times has now practically ceased to exist and only modern medicine is practised all over China.

Rural Medical Relief—The suggestions given by the Committee in regard to rural medical relief are neither practicable nor satisfactory. In order to increase the number of medical personnel for service in rural areas, the Committee has suggested that *hakims* and *vaids* who are at present practising in the villages should be given six months' training in medicine, surgery, midwifery, public health etc. By their estimate about 25,000 such practitioners can be trained within 5 years. Along with these, 3,000 out of 5,000 qualified indigenous medical practitioners can also be given the course and thereby made available for rural medical practice. The Committee accepts the fact that 25,000 *hakims* and *vaids*, who are to be given training, have never been trained in any of the existing institutions which impart instruction in indigenous systems of medicine. Most of them will be thoroughly illiterate. They may have a little knowledge of a few herbs which does not by any means make them fit for recognised practice of either indigenous or western systems of medicine. It would be an almost impossible task to impart instruction to these people on the subjects suggested by the Committee. Within six months these ignorant illiterate people will have to learn everything about first-aid, minor surgery which includes removal of foreign bodies, extraction of teeth, incision of wounds and abscesses, administration of saline, intravenous, subcutaneous and rectal, reduction of hernia, pregnancy and labour, gynaecological bleeding, principles of hygiene and preventive medicine, knowledge of communicable diseases, public health administration, preventive vaccination and inoculations, elements of vital statistics, maternity, infant and child welfare, school health work etc. etc. I agree that it will be possible to impart the instruction contained in the syllabus prescribed by the Committee to those who have passed out of recognised Ayurvedic and Unani schools of medicine. It will however be almost an impossible task to give this type of training to people who have had practically no sort of schooling. The question arises whether it is at all worthwhile even to train the 3,000 of the qualified practitioners which the Committee suggests for rural health work. If it is merely for the purpose of manning rural Ayurvedic dispensaries, these practitioners who have passed out of training institutions do not require any further training. On the other hand, if they are to be utilised for public health work in the villages, it will be a mere waste of human material. The type of public health work that is required in villages can certainly be done with equal efficiency by sanitary inspectors and health visitors, who can be given the necessary training within a period of six to nine months. On the other hand it will be a waste to utilise for the same work, practitioners of Indian medicine who have spent already three or four years in educational institutions for their special training. According to the scheme of health organization visualised by the Committee, primary units, secondary units and panchayat units should be mostly in charge of practitioners of Indian medicine. So far as the primary unit is concerned the Committee evidently is of opinion that the existing *vaid* or *hakim* in the village is competent enough, or perhaps will become competent enough after six months' training which they have advocated, to be put in charge of such

a unit. The Committee has worked on the erroneous assumption that something is better than nothing for the uncomplaining villager. They, however, conveniently forget the wiser adage 'a little knowledge is a dangerous thing' and that the untrained *vaid* when given the 'little knowledge' within the short period of six months will prove a positive menace to the village folk. It is not understood how the Committee can advocate such imperfect and dangerous experiments in the training of practitioners when they themselves have drawn attention to the grave possibility of inflicting injuries to patients by the use of drugs like penicillin and sulphanilamides etc., by indigenous practitioners without proper training. In my opinion the villager is as much entitled to get the best out of scientific medicine as a man living in a city. The suggestions made by the Committee for rural medical relief will not in any way solve the most difficult health problem which the country is faced with. The easiest and the quickest solution will be to train as many ancillary medical personnel as possible, of the category of sanitary inspectors, health visitors, nurses, *dais*, midwives etc., who will, under instructions from doctors be able to do most of the work that the medical men will otherwise have to do. It is only by increasing the ancillary medical personnel and establishing a large number of mobile dispensaries which can go to different villages, that the problem of rural medical relief will be solved. A doctor located in a health centre in a large village will be able to manage a group of villages in the surrounding area with the help of an adequate number of ancillary health personnel.

According to the scheme of health organisation visualised by the Committee, their primary, secondary, panchayat and tahsil units will necessarily be located in the rural areas. Estimated on the population basis suggested by the Committee 100,000 primary units, 30,000 secondary units, 6,000 panchayat units and 3,000 tahsil units will be required. As they have suggested that most of the rural health units should be in charge of *vaids* and *hakims*, who are given this short course of training, at least 136,000 such doctors will be necessary. As it is anticipated that 5,000 of these *hakims* and *vaids* will be trained every year, it will take nearly 27 years before the requisite number of medical personnel are made available. If the Committee's idea is to have for the whole of India, one system of synthesised medicine which, I expect, should be made equally available to both rural and urban population, I do not see how the bulk of the population of the country is going to be served with 'synthetic' medicine, if all the health units in the rural areas are to be manned for the next 27 years by *hakims* and *vaids*.

CASE NOTES

MULTIPLE AMOEBIC ABSCESS OF THE LUNGS

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D. N. N., aged 25, Hindu male, came with the following complaints: Cough and expectoration of foul-smelling sputum, haemoptysis, fever of irregular intermittent character, chest pain, loss of weight, weakness and loss of appetite. The symptoms started about a month ago. The onset was insidious with pain over the right shoulder, fever and unproductive cough. Later the cough became productive and sputum was purulent, foul-smelling and often contained blood. During the last 12 or 13 days he was having loose motions several times a day. The temperature ranged between 98°F and 102°F.

Past History—The patient was suffering from dyspeptic symptoms for the last one year and suffered from malarial fever about a year back.

Family History—No history of tuberculosis in the family.

On Examination—The patient showed moderate anaemia, a muddy complexion, emaciation (weight 86 lbs.) and slight oedema of the legs. Fingers showed second degree clubbing. Tongue moist and clean. No glands in the neck. Chest examination showed diminished movements at right base, impaired resonance on percussion over the right 2nd space and marked dullness over the right base below the 4th space in front, impaired resonance over the 2nd and 3rd space in front on the left. Breath sounds were diminished over the dull areas and crepitations were heard over the right base front and back. Cardiac apex was in 5th space on the left in the midclavicular line. The liver was enlarged 2 fingers below the costal arch and tender. The spleen was enlarged 1 finger below the costal arch and firm. Skiagram of the chest showed two cavities with fluid level and an irregular dense opacity at the right base. (See Plate).

Blood Count showed:

Hb.—70 per cent (Talqvist); R.B.C.—3·87 million.

W.B.C.—21,' 950; polymorphs—84 per cent, lymophocytes—8 per cent, monocytes—0 per cent, and eosinophiles—8 per cent.

A.T.—negative; C.T.—negative.

Sputum did not show any A.F.B. on repeated examinations.

A diagnosis of multiple lung abscess was made and the patient was put on a course of penicillin.

1,000,000 units of penicillin was given in 4 days. Then the patient started haemoptysis again. As the sputum appeared to be of an 'anchovy sauce' character, it was examined for the presence of amoebae. A fair number Ent. histolytica were seen in the sputum examined on 3 consecutive days. Examination of the fæces did not show any amœbæ, cystic or vegetative.

The patient was then put on emetine hydrochloride injections gr. i daily. The patient was staying in a private house and unfortunately, contrary to instruction he went to a lavatory at some distance. On the 2nd day while returning to bed, he suddenly had a syncopal attack and died immediately before any medical aid was available.

Discussion

Extra-intestinal amoebiasis occurs most commonly in the liver and less often in the lungs and brain. Reports of cases of pulmonary complications of amoebic liver abscess are met with fairly frequently. It has also been well recognised that pleuro-pulmonary symptoms and signs may be the most important and of primary significance in these cases. Dormer and recently Koszalka *et al*[3] described 10 cases in May, and Friedlander[1] reported 7 cases in 1941. Chaudhuri and Rai Chaudhuri[2] described two cases in 1946 and recently Koszalka *et al*[3] described 10 cases in May, 1949. According to one estimate pleuropulmonary complications occur in 15 per cent of patients having amoebic hepatitis or abscess.[4] Rogers[5] found a secondary abscess in the lungs post mortem in 26 out of 81 fatal cases of liver abscess in Calcutta, and in Europeans 20 per cent of liver abscesses opened through the lungs. In these cases the spread of infection is by extension through the diaphragm and the right basal pleura from the right lobe of the liver.

According to Manson-Bahr[6] there may also be a direct embolic spread of infection from the colon wall to the lungs. In these cases of primary pulmonary amoebiasis, there are multiple bronchopneumonic areas in the lungs.

The diagnosis of pulmonary amoebiasis is always difficult and is likely to be missed unless it is specially kept in mind. In cases of secondary pulmonary amoebiasis, the signs and symptoms are localised at the right base and evidences of liver disease, if present are of help. In primary pulmonary amoebiasis, the signs and symptoms are distributed widely in both lungs or may be localised in one area. As concomittant signs in the liver are not present, diagnosis is difficult. The presence of Ent. histolytica in the sputum, is not constant and therefore a negative finding is not of much value. Examination of fæces is also unreliable. The diagnosis in most cases is by exclusion and therapeutic trial.

In the present case, multiple lung abscesses were seen in the skiagram. The cause of these were in doubt. There was no obvious source of pyaemic embolisation of the lung. Response to penicillin was not satisfactory. These factors and the 'anchovy sauce' character of the sputum raised the suspicion and the sputum was examined for amoebae. Ent. histolytica was found in fair numbers on 3 consecutive days. The amoebae were identified by their movements and the presence of engulfed R.B.C. in their cytoplasm. Unfortunately the patient died suddenly, possibly of cardiac failure and therefore the result of therapeutic trial could not be observed. The patient had received only one grain of emetine hydrochloride. It is doubtful whether this can account for the sudden heart failure. Another possible cause of death is cerebral embolism. As the patient died instantaneously and no post mortem could be done, the cause of death will remain indefinite.

Summary

1. A case of multiple amœbic abscess of the lung has been described.

2. The diagnosis was confirmed by finding Ent. histolytica in the sputum.

3. The frequency and pathogenesis of pulmonary complication of amœbiasis are discussed.

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TRAUMATIC ANGIOSPASM

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Injury to arteries in fractures of long bones is not an uncommon occurrence. Brachial artery is the one which is most commonly involved and is usually associated with a supracondylar fracture of the humerus where the lower fragment has been markedly displaced backward. The injury is a contusion, an incomplete laceration or a complete crushing of the vessel. A more interesting type of case however is met with when the artery apperently remains almost untouched, but the periarterial vasoconstrictor fibres are stimulated by the violence which causes the fracture and by the displacement or subsequent manipulations so that a severe degree of spasm occurs in the vessels. This spasm may last for a long time and if unrelieved by surgical interference may lead to gangrene of the limb. The following case may serve as an illustration for this type of angiospasm.

Case Report

Miss K. Banerji, a healthy girl of twelve years, sustained a supracondylar fracture of the usual extension type with the lower fragment displaced backward. Examination two hours after the injury, showed considerable swelling of the elbow region, but the radial

pulse was present at the wrist. Radiological examination was advised. Four hours later the attending doctor reported that she had no pulse at the wrist on the affected side. On examination it was found that the limb had been supported on a right-angle splint at the X'ray clinic, and there was no radial pulse at the wrist. The lower part of the forearm and hand were cold and slightly numb. The splint was immediately taken off, and gentle friction was applied to the forearm with the hope of releasing any pressure on the vessel by the broken bone. This failed to give any relief. It was a late hour in the night, and the limb was kept swathed in cotton wool supported on a pillow with the elbow in extension with the hope that the spasm might pass off.

Nearly twenty-four hours passed in anxious waiting, but no sign of improvement appeared, and the œdema extended to the arm. An open operation was decided upon.

Under general anaesthesia an incision four inches long was made from the medial side of the arm, over the front of the elbow along the course of the brachial artery. The skin, superficial fascia and deep fascia were divided. There was hardly any bleeding from the cut tissues. The median nerve presented itself but brachial artery which is normally larger in diameter than the nerve was not visible. On gently lifting the nerve by an aneurism needle the artery was seen. There was no external sign of injury of the artery but it had undergone a condition of severe spasm so that its diameter was greatly reduced. There was no pulsation in it. To be sure that this was the brachial artery it was traced down to its bifurcation. These two main branches were also pulseless. In following the artery downwards the loose areolar tissue around it had been dissected off. When the whole artery had thus been cleaned throughout the length of the incision it suddenly and quite unexpectedly began to pulsate, and pulsation also reappeared in the radial and ulnar arteries. Fine arterial twigs which had been divided and which had not bled at all, now began to spurt and required to be ligated. Pulse at the wrist however did not appear. But now that circulation had returned in the brachial and upper part of the radial arteries it was hoped that circulation would be restored in the lower forearm and hand.

Now turning to the fracture it was found that the lower end of the upper fragment of the humerus had a very sharp edge and the medial part of the brachialis muscle was clean cut across as if with a razor. Attempt was now made to reduce the fracture, but curiously enough, on applying long axis traction to the arm, the brachial artey again ceased to pulsate. Traction was released and it took several minutes for the pulsation to reappear in the artey. Two attempts were made and the same phenomenon was observed. It was now decided to give up the idea of reducing the fracture for the present. The wound was closed in layers and the limb was put up in plaster in right-angled flexion at the elbow.

Radial pulse had appeared the next morning, and the hand and fingers were warm. On the seventh post-operative day the stitches were removed. It was now decided to reduce the fracture under general anaesthesia, and under the fluoroscopic screen. The patient was under the anaesthetic and no sooner was traction applied than the pulse disappeared from the wrist on the affected side and did not reappear before another three or four minutes, though the radial pulse remained unaffected on the other side all this time. A second attempt was made after ten minutes with exactly the same result. Further attempt to reduce the fracture was given up, and the limb was put up in flexion at 90° in Plaster of Paris.

The idea of any further manipulation or reduction of the fracture was given up, and the fracture united in bad position. Subsequently however when the fracture had united, active or passive movements made at the elbow did not affect the circulation in the brachial or radial arteries, and gradually a fair degree of movement was possible at the elbow.

SUMMARY

A case of supracondylar fracture with posterior displacement of the lower fragment is described.

The radial artery becomes pulseless on the affected side due to manipulations subsequent to injury and remained so until relieved by operation 24 hours later.

At operation the artery was seen uninjured but thrown into a spasm, its diameter reduced to one-third its normal size.

On removal of the periarterial loose areolar tissue and incidentally its sympathic plexus, pulsation reappeared.

Subsequent attempts at reduction of the fracture threw the artery again into spasm, due probably to drag on fine filaments of the sympathetic nerve fibres.

After the fracture had united, forcible movement at the elbow no longer caused any spasm of the brachial artery—probably there being no drag on the fine peri-arterial sympathetic twigs.

FRACTURE PATELLA WITH DISCHARGING SINUS

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Mr. P. Appalaraju, aged 72 had a fall and fractured his left patella some six months before he came for treatment. A few days after the fracture it went septic and a big abscess formed over the knee. The abscess was opened by a local practitioner and it healed leaving a discharging sinus. The patient could only just move about with the help of a walking stick.

On 17-12-'47 when he was operated on, the skin over the knee was healthy except for the discharging sinus. The broken ends of the patella were 1½" apart. The knee joint was quite healthy. The patella was removed. The sinus was excised in its entirety. The torn ends of the quadriceps tendon were trimmed and sutured together. The outer half of the tendon

was involved in the original abscess and the sinus thereafter and so, was very fragile and inelastic. Only the medial half of the tendon could be sutured together and one had to be contented with bringing the outer half as close as possible. The wound was closed with intubation for penicillin instillation and plaster of Paris cast applied from mid-thigh to the foot.

3 c.cs. of a 500 units per c.c. solution of penicillin was instilled through the tube thrice daily for five days and 15000 units penicillin three hourly was given parenterally for seven days. The penicillin tube was removed on the 6th day. The temperature kept normal from the 7th day onwards. Sutures were removed on the 12th day and the healing was perfect.

Regular quadriceps drill was started from the day sutures were removed and gradually intensified.

After fifteen days non-weight-bearing exercises were started.

Ten days after this the patient was allowed to stand up. Soon the patient began to walk with a little help and on 30-1-'48, roughly one month and fifteen days after the operation the patient walked out of the Nursing Home without help, though with some limp. Now he is getting about his agricultural work without any difficulty.

In spite of his age and the presence of sepsis, penicillin and exercise restored his knee to normal.

COMPOUND FRACTURE—PRIMARY SUTURE 20 HOURS AFTER INJURY

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I. J. Raju, aged 14 yrs. fell from a tree at 4 p.m. on 14-7-'48 and landed on his hands with the arms stretched. Simple Colles' fracture of left radius and compound fracture of both bones of right fore-arm resulted. The right radius was separated at its lower epiphysis and the ulna was fractured a little above its lower epiphysis. The upper fragments of both the bones stuck out of the skin above the wrist to an extent of about 1½". The ground where he fell was wet and the bone ends stuck into it.

At 12 noon on 15-7-'48, 20 hrs. after the injury, the boy was operated upon. The wound was freely excised, the bone ends were throughly cleaned and the fractures reduced. The wound was closed with intubation for penicillin instillation and the forearm put up in plaster of Paris.

3 c.cs. of a 500 units per c.c. solution of penicillin was instilled into the wound thrice daily and parentaral penicillin 15000 units every three hours was given for five days. The penicillin tube was removed on the sixth day. The temperature was normal from the fourth day onwards and the boy was then allowed to get up. Sutures were removed on the 10th day and the wound healed by first intention. The plaster of Paris was removed after four weeks and the fractures were found to have united satisfactorily.

A compound fracture of both bones of the forearm should be immobilized for at least six weeks.—ED., *J. Indian M. A.*

POLIOMYELITIS WITH BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

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INTRODUCTION

In November, 1947, acute anterior poliomyelitis broke out in an epidemic form in Carnicobar Island (Bay of Bengal). It broke out all on a sudden almost throughout the entire expansion of the island and took a heavy toll of the adult population. There were in all 976 attacks out of a population of a little below 10,000; of this again about 209 died and a large number became crippled. I had the privilege to study the epidemic from the beginning to the end. There was a large clinical variety of different poliomyelitis cases—spinal, bulbar, midbrain, encephalitic, cerebellar, ascending spinal, descending spinal and mixed in various combinations.

CASE REPORTS

Case 1 is one of these cases occurring at the height of the epidemic. I studied the second case in the R. G. Kar Medical College Hospital, Calcutta.

Case 1—Panja, male, 24 years old, admitted on the 3-12-47 with (1) Fever—2 days. (2) Headache—2 days. (3) Pain in the throat—1 day. (4) Difficulty of deglutition with nasal regurgitation of water—1 day.

Onset—Two days ago, in the evening the patient had some rise of temperature with headache, pain in the whole body, diminished appetite and some nausea. The next day the temperature increased and with that all his previous complaints aggravated. He also felt difficulty of swallowing food and also had nasal regurgitation of water.

General survey—A young adult of robust constitution looking very ill with temperature 102°F, drowsy, sweating, prominent neck veins, swelling of the supraclavicular fossae of both sides, inspiratory and expiratory stridor, chest held in position of extreme inspiration—intercostal spaces bulging out and extreme expiratory difficulty—pulse 120 p.m. B.P.—S/D 130/86 m.m.

Nervous system—Husky voice, pupils widely dilated but reacting to light. Paralysis of the right side of the face, paralysis of the soft palate and the pharynx. Stiffness of the neck and the back. Babinski's sign—Negative both sides. No sensory change. Laryngoscopic examination of the patient could not be done.

Respiratory system—Deeply cyanosed, subcutaneous emphysema in the supraclavicular fossae bubbling rales and rattles throughout the lungs.

Heart sound—distant and almost drowned in the moist sounds in the lungs.

About an hour later, cyanosis intensified so much so that the tongue became deep blue, subcutaneous emphysema extended, the chest became greatly emphysematous and the patient died at the height of this.

Case 2—Brojendramohan De, Hindu, male, 15 years old, was admitted on the 20-7-48 at 6-50 p.m. with.

(1) Continued fever—7 days.
(2) Extreme paresis of the lower limbs—7 days.
(3) Paresis of the upper limbs—6 days.
(4) Difficulty of breathing—since morning.

Onset—Seven days ago, the patient had slight rise of temperature (99°F) with vague pain in the lower limbs. The next day he felt weakness in the lower limbs and had difficulty in walking and in starting micturition and had a rise of temperature to 101°F. The same day in the evening he felt pain and weakness of the upper limbs, lost all power of movement of the lower limbs and had retention of urine. On the following few days the temperature came down and ranged between (98·4°F to 99·4°F) and also with this he regained some power to move the limbs also the bladder function returned. On the 6th day, the patient again had rise of temperature to 101°F but other features were the same. The next morning, the patient was found to have some difficulty of breathing.

General survey—A boy of good nutrition, looking ill with scalenii and alæ nasi moving and epigastrium sinking in during insoiration. Pulse—112 p.m. Resp. 26 p.m. B.P. 110/70 mm. of Hg.

Nervous system—Intellectual function—slightly dulled. He was straining to speak but the voice was husky. The eyes were congested, pupils dilated but reacting to light; paresis of the right side of the face; palate did not move on the right side; no nasal regurgitation of water taken slowly. All muscles of the limbs were hypotonic. Jerks—absent in the lower limbs and sluggish in the upper limbs (only Biceps and Triceps). No sensory disturbances.

Alimentary system—Tongue—thickly coated and flabby. Abdomen—slightly tympanitic. Liver—slightly palpable. Bladder—empty.

Cardiovascular system—Slight cyanosis, no apparent cardiac enlargement. Impulse—thumping; heart sounds—loud.

Respiratory system—Entirely thoracic type of respiration; indrawing of the lower intercostal spaces during inspiration; there was no adventitious sounds in the lungs.

Laboratory examination—Hb.—100 per cent. R. B. C.—5·2 mill/c.mm., W.B.C.—13,400 c.mm. Poly—80 per cent, lympho—16 per cent, eosino—2 per cent, mono—2 per cent, parasites—not found.

The next morning—Temperature 99°F., Pulse—98 p.m. Respiration 32 p.m.

The patient was stuporose; difficulty to speak had increased and the respiratory difficulty was greater. The eye balls were prominent and congested; pupils dilated but reacting to light. Slight cyanosis apparent in the nail-beds. The other features were same.

Three hours after this—The features of the case rapidly deteriorated. The patient had a low muttering delirium. Difficulty of respiration was marked. Respiratory stridor was present (both inspiratory and expiratory). The patient was semiconscious, eyes greatly congested, pupils dilated but reacting, neck veins congested. The accessory muscles of inspiration vigorously acting with indrawing of the intercostal spaces and epigastrium. Other features the same.

While we were still standing by the bed-side the patient had progressive slowing of respiration and became increasingly cyanosed. Suddenly his respiration stopped at the beginning of expiration, the abdominal muscles vigorously acted, the epigastrium puffed up and the intercostal spaces bulged out, the patient had convulsion restricted to the tongue and jaw; the tongue became almost blue black, the face greatly suffused, eye balls protruded out, pupils widely dilated, neck veins stood out greatly congested. At this stage the patient died.

Discussions

In the case of the first patient the diagnosis of bulbar form of poliomyelitis was hardly in doubt as the case occurred at the height of the epidemic. It is really a fact that a case coming in with a history of rise of temperature with general malaise, headache and stiffness of the neck followed in the next twenty-four hours by development of facial, palatal, and pharyngeal palsy is almost certainly one of acute bulbar poliomyelitis unless otherwise proved. There was no cause for thinking it to be diphtheritic—the patient being a young adult, the palsy coming in within twenty-four hours of the fever and especially because the throat looked perfectly healthy not to speak of the presence of any membrane.

Diagnosis of poliomyelitis in the second case was not much difficult. The patient had rise of temperature with headache, general malaise, vague pain in the lower limbs followed in the next twenty-four hours by paresis of the lower limbs and bladder disturbances. This was soon succeeded by complete flaccid paralysis of the lower limbs and retention of urine. With such a clinical picture myelitis comes to our mind first as this is the commonest neurological disease we meet with in our hospitals. But sensory changes were conspicuous by their absence.

The paralysis took an ascending course and so it is relevant to bring into consideration the ascending paralysis of Landry. But this disease is generally afebrile, leucocytosis is not a feature and the course is never intermittent. Whenever the progress stops rapid improvement follows. There were many peculiar features in this case—although these are not unique. The course of the disease in this case was intermittent. From the second day the temperature subsided, paralysis of the toes improved and also the bladder function returned. But suddenly on the sixth day the temperature shot up and the progress started anew—affecting the diaphgram, lower intercostal muscles and the bulb.

Then again the upward march of the paralysis was scattered. The abdominal and upper intercostal muscles were spared and the more resistant phrenic nerve centre was affected leaving aside the more readily susceptible intercostal nerve centres.

The most interesting feature in both the cases (for which especially these two cases are being reported) is the common mode of termination in both.

The first case was admitted with acute respiratory difficulty—both inspiration and expiration being difficult, associated with laryngeal stridor both in

inspiration and expiration and subcutaneous emphysema in the supra-clavicular fossae. These were all possibly due to paralysis of the abductor muscles of the larynx which obstructed passage of the air out of the lungs during expiration. The result of this was interstitial emphysema in the lungs. The liberated air then travelled beneath the pretrachial fascia and manifested as subcutaneous emphysema in the neck.

In the second case, the patient had difficulty of breathing with husky voice on the 6th day. The next morning, he was found to have increasing difficulty of respiration with indrawing of the intercostal muscles during inspiration and laryngeal stridor both in inspiration and expiration indicating some obstruction in the larynx. Within a short time of this, frequency of respiration progressively slowed down due to progressive failure of the respiratory centre. All on a sudden the respiration stopped and the last phase was one of acute respiratory obstruction. This was possibly due to bilateral abductor paralysis of the larynx shutting up the airway to the lungs.

We know that the intrinsic muscles of the larynx are supplied by the recurrent laryngeal nerve excepting the cricothyroid muscle which is supplied by the external laryngeal nerve. The fibres of both originate from the lower part of the nucleus Ambignus in the medulla (i.e., the cerebral centre of the 11th cranial nerve) and the peripheral part is distributed through the vagus nerve (of which both the recurrent laryngeal and the external laryngeal nerves are branches). "The original function of the laryngeal muscles is that of a sphincter to prevent entrance of fluid into the lungs and this sphincter or adductor is the only muscle in the larynx of the primitive air breathing animals; the abductors are a later addition to assist entry of air." Therefore, in a progressive lesion the more recently acquired function of abduction is affected first while the primitive function of adduction is more resistant and sustains longer. In both the cases there was progressive affection of the bulbar motor nuclei, and so the abductor muscles were paralysed before the adductors.

In the first case, the abductor muscles were already paralysed before admission and the abductors closed the larynx; so the patient died of asphyxia and heart failure.

In the second case the laryngeal muscles were already affected before admission as evidenced by the huskiness of the voice. In the last phase abductors were completely paralysed when the antagonistic abductors sheet up the glottis and closed the picture.

ACKNOWLEDGMENT

I am grateful to Dr. J. C. Gupta, M.B. (Cal.), M.D. (Cologne), Visiting Physician and to Dr. K. C. Sarbadhikari, M.B. (Cal.), F.R.C.S. (Edin.), F.R.C.S. (Eng.), Superintendent, R. G. Kar Medical College Hospitals for permitting me to publish the second case.

I am also grateful to Lt. Col. C. C. Kapila, O.C., Central Military Pathological Laboratory, Poona, Officer on special duty at the Nicobar Epidemic for permitting me to publish any case observed at the Epidemic.

XXVI ALL-INDIA MEDICAL CONFERENCE, ALLAHABAD

OFFICE—*Allahabad Medical Association, 5-A, Elgin Road, Allahabad.*

THE 26th session of the All-India Medical Conference will be held at Allahabad in the University Senate Hall on 27th, 28th and 29th December, 1949.

There will be a Scientific and Medical Exhibition, Scientific Session and an Exhibition of Commercial Medical and Surgical Products.

The Reception Committee and the Allahabad Medical Association have the pleasure to extend their invitation to colleagues all over the country to attend the Conference in large numbers and make it a success befitting its all-India character.

Members of the profession, specially those on the teaching staff of medical institutions and others who have access to or possess clinical material or aptitude for research are requested to liberally contribute papers and to Symposia for the Scientific Section and thus serve the best interests of the profession and the country.

The subjects for the Symposia are:—

1. Epidemic dropsy,
2. Role of Antibiotics with special reference to Tuberculosis,
3. Cirrhosis Liver in Infants,
4. Surgical Dyspepsias,
5. Uterine Hæmorrhage.

As accommodation is rather difficult to obtain in Allahabad these days, members of the profession, delegates and others who intend to attend the Conference should send their names with addresses by the 31st of October, 1949, indicating if they would like to have accommodation in any Hotel.

JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION
CALCUTTA, SEPTEMBER, 1949

MEDICAL EDUCATION AND RECIPROCITY

The last great war has shown that no country can afford to remain isolated and it has got to make a pact or alliance for its defence and other purposes. As a result, we have now the U.N.O., the Council of Europe, the World Health Organisation and the World Medical Association. Previously no country would allow any one, who is not its own national, to practise medicine, no matter how well-qualified he is, unless he became a domicile or acquired a qualification in that country. A person qualified in the British Commonwealth could only practise in the different units of the British Commonwealth and nowhere else. But now ideas are fast changing and one ought to have a new and broader outlook on the problem. U.N.O. has taken up the work of rendering medical assistance in different parts of the World and the time has come when some of the barriers placed in the way of doctors from one country rendering medical aid in another are revised.

The Indian Medical Council was constituted by an enactment in 1933 and contained a clause which empowered it to enter into reciprocal negotiation with other countries. But in actual practice except with Great Britain there was practically no reciprocity in the real sense, as even some of the colonies refused to enter into direct reciprocity unless through the General Medical Council of Great Britain. This was derogatory to the self-respect of India and was soon abandoned. But it was felt that some exception should be made in case of Indian nationals who had received foreign medical degrees of repute.

Now with the attainment of independence it is quite natural that India should not allow any doctor from a country to practise in India which does not allow Indian doctors to practise there. This tendency is bound to create complications and difficulties in the matter of medical aid which India is seeking to make up its deficiency by asking the aid of World Health Organisation or U.N.O.

India is now sending a large number of post-graduate medical students to foreign countries every year and spends a very large amount of money (nearly 3 crores of rupees) but even then one is not so sure if they get the full opportunities of practical training. It has also become evident that by sending a few scholars scattered over various centres, one would not get the desired result. A surgeon, physician, radiologist, pathologist, etc., however efficient, cannot produce as good results individually as a team or unit of them could do. Moreover people trained at different centres, under different methods and environments, when put together, might not be able to work as a team very efficiently. It would perhaps be better if a unit of workers could be sent to each large centre, where they could have comprehensive and thorough training in the various departments as one team and on their return could be posted as a complete team or unit in one institution.

The other alternative which has been suggested in certain quarters is to invite on a short and long term basis distinguished medical men or teams from abroad. They will help to fill up the gap in certain directions particularly in the matter of post-graduate medical education, research, and in some aspects of medicine and surgery, *e.g.*, surgery of heart, lung and brain, microbiology, etc. It has got to be decided whether it would not be better and cheaper in the long run to import these doctors or a traveling medical team from foreign countries even if there is no reciprocity with those countries in the larger interest of India.

These workers would work in Indian conditions and surroundings and train a large number of Indian medical graduates in those specialities much better than a post-graduate student could do abroad.

The Indian Medical Council Act will probably be amended shortly by the Government. We draw the attention of the Government and the profession to consider this aspect of the question dispassionately as to whether those foreign doctors, with whose countries there is no reciprocity and who are experts in their own line, whose qualifications are recommended by the Provincial Medical Councils and subsequently again scrutinised by the Examination Committee of the Indian Medical Council, who are rendering good and humanitarian services to India, could be given some temporary registration and in case of Indian nationals permanent recognition.

EPIDEMIC DROPSY

Of late, the citizens of Calcutta, are becoming increasingly panicky owing to the declaration by the Health Authorities of the Government of West Bengal that epidemic dropsy has broken out in the city and suburbs, particularly Howrah, in an epidemic form.

In fact the epidemic may be said to have started at Howrah about a year or more ago, where a fairly large number of cases were detected by the doctors of the locality and traced to sources of contaminated mustard oil depots which were the common sources of supply of the persons who were the earliest victims of the disease.

For the last six months or more the incidence of the disease has been rising slowly but steadily. The Health Authorities of the Government as well as those of the Calcutta Corporation failed to evince any noteworthy enthusiasm in this connection in their attempt to tackle this serious problem in a proper and befitting manner and to stop the adulteration at its very source of supply. The result of it has already made itself felt in the fact that incidence as well as mortality from epidemic dropsy, rising from week to week are being reported in the papers and are being corroborated by the informations collected by members of the Calcutta Branch of the I.M.A.

It may be recalled here that the first outbreak of epidemic dropsy occurred in Calcutta in 1877 and the name of the disease was given by McLeod.

Although, the disease is still considered to be one of uncertain ætiology with its own peculiar host of symptoms (characterised by œdema especially of the lower extremities towards the close of the day, with a peculiar blotchy erythema of the skin with a varying range of pyrexia, cardiac symptoms, glaucoma, hæmorrhages, cutaneous pigmentation and a nodular eruption)—recent observations on feeding experiments in volunteers and on repeated outbreaks of the disease in a closely observed small community, indicate that mustard oil, more than anything else, as an article of diet has an important causative role to play. The observations of Chopra, Pasricha and Lal go to lend further support to the theory that it is not the mustard oil itself but an adulterant, namely, the oil derived from the seeds of Argemone mexicana—the seeds of which very much resemble mustard seeds in appearance. Certain consignments of mustard oil which are adulterated with the oil derived from the seeds of these wild and luxuriantly growing yellow-flowered thorny weeds,—either deliberately for the sake of profit,—or what is far less likely—accidentally —while harvesting the mustard crop from fields where these weeds also grow side by side,—have been traced as the common source of supply for a bunch of families many or most of whose members fell victims to the disease. The weed is popularly known as Kata-Kar, Sial-Kanta, Odissimari, in different parts of the country. Their seeds resemble closely and are inseparable from the black variety of mustard seeds.

Epidemic dropsy as a clinical entity is distinguished from beri-beri by several features. It is endemic or epidemic in certain areas. It does not occur in infants, peripheral neuritis is absent, the œdema is associated with a blotchy erythema and a slight thickening of the skin along with cutaneous nodules; there are not infrequently although not invariably a remittent or intermittent type of fever, gastro-intestinal disturbances, glaucoma, and hæmorrhages into the retina or from the mucous membranes and from piles when present. Abortion is a frequent feature in pregnant women. The adulterated mustard oil, responsible for the causation of the disease gives a positive colour reaction with nitric acid. But it is considered that the fraction of the argemone oil which gives the reaction is not responsible for the disease. The argemone oil is rendered non-toxic when heated to a particular temperature. The adulteration may also be detected by another more delicate test,—the reagent employed being ferric chloride.

The pathological study on epidemic dropsy has not as yet been very extensive owing to dearth of autopsy materials and their careful post mortem examination. We may draw the attention of our readers to the original paper published on the subject in the *Calcutta Medical Journal* (April 1940) by De and Tribedi entitled "Observations on the pathology of Epidemic Dropsy" which practically incorporates all the pathological informations available on the subject.

In spite of what has been said above, the pathogenesis of the disease is still obscure. De and Tribedi have advanced the suggestion that the disease is comparable to many virus diseases in which we find that a certain virus exercises a particular predilection for a particular type of tissue leaving others in tact, such as neurotropic, dermotropic virus etc. Not unlikely, they say,—that epidemic dropsy poison is vaso- or angiotropic in nature. We draw serious attention of our clinicians and pathologists to utilise the clinical and autopsy materials offered by the prevailing epidemic so that they may form themselves into a team work of study and add to the fund of world knowledge.

The theory of mustard oil is not only attractive but is fairly well grounded. It has been the opinion of authoritative observers that feeding of volunteers with diseased rice has failed to produce symptoms of the disease whereas feeding of volunteers with the suspected specimen of oil has successfully produced some of the essential features of the disease such as fever and œdema of the legs with erythema. A biopsy from the affected area of skin has demonstrated some degree of capillary dilatation in the deeper layers of the skin. It is quite possible that the argemone oil might give rise to a poisonous effect which might lead to a vasodilatation either by its irritant effect or by liberating histamine from the tissues in some way which has not yet been fully explained or understood. The unexplained features of the disease are—its seasonal incidence, epidemiological cycles and peculiarities, the chronicity of its clinical picture and repeated relapses in spite of the withdrawal of mustard oil from diet. All these have to be considered carefully and explained.

Even accepting mustard oil as the cause,—it has been questioned whether it can be due to any other reason that the mere presence of argemone oil in the same. For example, may it be due to any contamination of the oil with any other poisonous matter such as decomposed glucosides? Mustard seeds when stored for a long time develop moulds and fungi,—and glucosides are known to be easily decomposed to toxic substancs by the action of moulds and fungi. The nitric acid test may yield similar positive reaction (comparable to the argemone test) in the presence of any alkaloid or broken down glucoside. It has not as yet been definitely established as to which particular constituent in the argemone seed is responsible for the detection test.

The Government should enact immediate legislation to authorise seizures and confiscation of all samples or consignments of mustard oils or mustard seeds which show evidence of contamination, and to inflict deterrent punishment to persons who are responsible for manufacture and supply of such contaminated stuff.

The Governments of all those provinces that are the main growers of mustard seeds must exercise a close vigilance over the cultivation of mustard plant so that contamination may be completely prevented. We owe it to rumour that even cultivation of argamone is undertaken on a wide scale for the purpose of mixing their seeds with mustard seeds. Close control by the Public Health administrators of the city, must be enforced on the quality of mustard oil or seeds imported into the province.

The Calcutta Corporation and the Public Health laboratories of the Government of Bengal ought to offer free facilities to the general public for having samples of mustard oil tested for adulteration.

We draw the attention of all concerned to this grave menace to the health of the citizens. The injury caused by the disease is usually insidious and slow, so that the chronicity and its far-reaching damage and debilitating effects fail to attract the serious attention they deserve.

NOTES AND NEWS

SPREAD OF INFANTILE PARALYSIS IN DIFFERENT PARTS OF INDIA

Though no definite statistics are available, the Central Health Ministry Official circles believe that the increasing incidence of poliomyelitis (infantile paralysis) in different parts of India warrants immediate action to check the disease.

It was at their suggestion that the regional office of the World Health Organization in Delhi asked its HQ in Geneva to send as soon as possible 20 portable respirators (iron lungs) to India for use in the treatment of emergency cases of the disease.

The Directorate of Health Services, Government of India, has been asked to inform the regional office of electrical specifications such as to the voltage and the type of current which the directorate would like the respirators to satisfy.

The first consignment of iron lungs will be flown to India as soon as information regarding specifications is communicated to manufacturing firms in the U.S.A.

The portable respirators is made largely of rubber, each costing $1,000 in the U.S.A. To obviate the difficulty created by India's dollar shortage, the Central Health Ministry has suggested the creation of a pool of iron lungs for the South-East Asia region. If such a pool is created, WHO's member States will receive, on a loan basis, respirators from this pool for use in times of emergency. The matter is now under negotiation.

Other Measures—The Central Health Ministry has planned several other measures to combat the disease. In order to obtain quick information about incidence, it has been suggested that the disease be declared as notifiable under the Epidemic Diseases Act. The Delhi administration has been directed to take the step immediately and it is hoped that Provincial and State Governments will follow suit.

Instructions are being issued to doctors to undertake systematic investigation into factors on which the disease thrives and into the methods of prevention. The Ministry proposes to develop regular research units in Bombay for this purpose.

Dr. K. C. K. E. Raja, Director-General of Health Services, will soon visit Bombay and Calcutta to discuss with the Provincial Governments the situation arising out of the outbreak of the disease at these places.

According to reports received at the directorate, five cases of attacks of poliomyelitis have so far been recorded in Delhi. Local hospitals are being provided with about 30 beds to accommodate patients suffering from the disease. Seven nurses are being given special training for attending to polio cases.—*The Statesman*, 29-8-49.

INDIA AND *WHO*

An article has recently appeared in People's Health, Madras (1948, 3, 57) in which an anonymous author, described by the editor as a "high authority", gives a history of WHO and explains the advantages which India may obtain from its participation in WHO.

"..... India's role" it is stated, "is twofold, firstly as a 'donor' and secondly as a 'recipient' .... Indian health services are still .... backward .... She has, however, an ample amount of local talent in individual fields of medical endeavour. This talent is being placed at the service of WHO through eminent Indian experts. No less than five Expert Committees of the WHO contain Indian members who are taking a very active part in the formulation of WHO policy and programmes. In the control of epidemic and endemic diseases, India has a particularly important part to play on account of the extensive field experience gained in the country .... In the field of medical research too, Indian scientists have played no mean part ... Even to-day Indian medical institutions are being utilised for the training of health personnel for other countries of Asia ...."

"As a 'recipient' country India has, of course, much to gain from her association with the WHO in the strengthening of her national health services by helping to train a vast army of doctors, nurses and midwives, health visitors and sanitary inspectors .... Fellowships for postgraduate studies in foreign countries are available from the WHO and eminent foreign experts in different medical specialities can be brought here if desired for demonstrating to the medical profession in India the modern advances in scientific knowledge. A certain amount of medical equipment and literature is also available. The main feature of WHO assistance however is the sending of demonstration and training teams to different countries .... These teams consist of experts who are posted to member countries for short periods in order to demonstrate disease control in selected areas and to train local personnel who will then take over the work and continue it under the national health authorities.

"As the largest partner in the South East Asia region," concludes the author, "India also has to lead the way in the energetic fulfilment of these international rights and obligations and so to strengthen further her ties with the WHO which is perhaps the only international organisation free from political con-

siderations, determined only to raise the standards of health of all peoples without distinction of race, religion, political belief, economic or social conditions." *Chronicle World Health Organisation*, **3**:95, 1949.

U. N. AID TO INDIAN CHILDREN

The United Nations International Children's Emergency Fund is carrying out a 750,000-dollar programme of aid to needy children in India, it was announced in Lake Success on 26th August last.

A 250,000-dollar programe on similar lines for the children of Pakistan is being set up.

The programme for India covers five fields: An anti-tuberculosis campaign, malaria control projects, scholarships for doctors working in the field of child welfare, a health education campaign and demonstration of child feeding projects.—*P.T.I—R.*

UNITED NATIONS EDUCATIONAL, SCIENTIFIC CULTURAL ORGANISATION

UNESCO proposes to publish later in the year a Manual on the International Exchange of Publications.

It is intended to publish as an annex to this manual a classified list of institutions, including universities, scientific institutions, learned societies, etc., throughout the world, which are willing to exchange either their own publications or other publications which they have regularly at their disposal. In the course of its activities UNESCO has been able to obtain a considerable amount of information concerning the availability of exchange material, but we feel that the information at our disposal is still far from complete. All institutions which have so far not sent to UNESCO details of their exchange material in one form or another are therefore urged immediately to communicate the following information to the UNESCO Clearing House for Publications, 19 Avenue Kléber, 16*e*:

(*a*) Name and full address of institution.

(*b*) Exact titles of publications offered. (In the case of duplicates offered for exchange purposes actual lists of duplicates are not required, but only a statement that lists of duplicates are available. Institutions possessing a catalogue of their own publications available for exchange are asked only to send a copy of the catalogue or to give a full bibliographical description of the catalogue).

(*c*) Institutions which wish to exchange their publications only under certain conditions, are asked to state what these conditions are.

Only information which reaches Unesco before October 1st, 1949 can be used in the Manual and it is therefore in the interests of all institutions concerned to communicate immediately with the above address.

INSTITUTE OF DENTAL SURGERY

The Institute of Dental Surgery has been established by the Ministry of Health, United Kingdom, at the Eastman Dental Clinic, Royal Free Hospital, Gray's Inn Road, King's Cross, London, for postgraduate instruction to dental practitioners who are on the dental register of any country in the world. This institute of Dental Surgery with its Committee of Management and academic staff intend to carry out postgraduate work and research for the primary purpose of training teachers and specialists in the various branches of dental surgery. Research will be carried out in each department and stress will be laid on preventive dentistry and caries control subjects which have received little attention in the past. The American Congress has appropriated $1,000,000 to be used in establishing a topical fluoride demonstration unit in each state. Plans for the demonstration were worked out at a conference of state and territorial dental health directors with the Public Health Service and the Children's Bureau in June, 1948. Actual work will begin in the very near future. Of course, these demonstrations are carried on in co-operation with state health agencies and with state and local dental societies.—*J. All-India, Dental A.*, **21**:131, 1949.

SYNTHESIS OF CHLOROMYCETIN

The Parke Davis Research Laboratories announce that a team headed by Dr. Leon A. Sweet, director of the research department of Parke, Davis & Co., has synthesised the antibiotic chloramphenicol ('Chloromycetin'). Production on a large scale is being undertaken; and thus, for the first time, an antibiotic is being manufactured for use. Chloromycetin has given promising results in a number of infections, including scrub-typhus, typhoid fever, Rocky Mountain spotted fever, and undulant fever.—*Lancet*, **1**:576, 1949.

AMERICAN MEDICAL DEVELOPMENTS IN 1948

Albert Deutsch from *Mademoiselle* writes:—Sober American scientists who disdained overdramatic claims came out of their laboratories, clinics and operating rooms this past year with solid contributions to the diagnosis, treatment and prevention of diseases. Two new drugs that may untimately rival sulfonamides, penicillin and streptomycin in healing power made clinical debut in 1948. A number of promising leads to the conquest of defiant diseases were explored, and significant new diagnostic tools and surgical procedure were successfully developed.

The terrible toll of America's number-one killer, heart disease, was whittled down by several important discoveries. Dr. Harry Gold of Cornell University Medical School disclosed a new drug-and-diet treatment for congestive heart failure. One out of four victims of this condition used to die; the new treatment cuts the death rate to one in 10. Further, it

gets results in six days, as compared with 18 days required by previous methods. The new treatment combines administration of dixitoxin and a mercurial diuretic, plus a milk-and-water diet for a certain period. It greatly reduces the danger of such complications as coronary thrombosis.

The menace of coronary thrombosis itself was greatly lessened. A special committee set up to study the effect of anticoagulant drugs on coronary thrombosis complicated with myocardial infarction (reduced circulation in the heart muscle resulting from coagulation) rendered its report on the basis of 800 carefully studied cases. The committee declared that when anticoagulant drugs—dicumarol or heparin or both in combination were used in treating coronary thrombosis with myocardial infarction, the death rate fell from 23 to 13 per cent. Added impetus was given to the fight against heart ailments when the American Congress in 1948 created the National Heart Institute to expand and co-ordinate research in this field.

New radioactive weapons drawn from atomic furnaces opened new sectors in the fight on cancer, number two among death-dealing diseases in the United States. As valuable by-products of the atomic piles, increasing amounts of radio-isotopes became available for use in research, diagnosis and treatment of cancer and other afflictions of man. Through radio-isotopic tracers, it was possible to tag chemicals and with the aid of Geiger counters to observe their course as they passed through the blood stream, digestive organs and other parts of the body. The isotopic traced has become the most important investigative tool yet devised for exploring the complex economy of the human body in health and disease.

Dr. John H. Lawrence of the University of California Radiation Laboratory reported that radio-active phosphorus obtained from cyclotrons and atomic ovens had given "a nearly normal life expectancy" to victims of polycythemia vera, a cancerous condition caused by uncontrolled proliferation of red blood cells. Radioactive phosphorus continue to be used cautiously for certain types of leukemia—a cancerlike disease of the blood. It does not cure, but it seems to retard the course of the fatal disease in some cases. Radio-active iodine was used, with similarly modest results, in treating some cases of thyroid cancer.

When the great American baseball hero, Babe Ruth, died of a throat cancer in the summer of 1948, it was publicly revealed that his suffering had been eased and his life perhaps prolonged by a new drug, teropterin, a folic acid derivative that can be derived from spinach. Folic acid, already proved effective in the treatment of pernicious anaemia, has yet to demonstrate its therapeutic value in cancer.

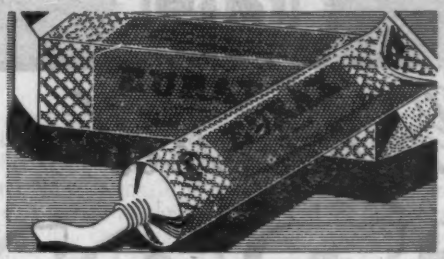
Three doctors at the Johns Hopkins University radiology department reported that they had developed a new mass X-ray test for cancer of the digestive system, which affects 100,000 Americans each year and kills four out of every five. The new test reduces the cost, time, danger and pain attending former tests for stomach and other digestive-tract cancers. It may make possible the detection and prompt treatment of thousands of cases which otherwise would terminate fatally. Some cancer experts, however, have expressed skepticism about applying the Johns Hopkins X-ray test on a mass basis. By and large, surgery and radiation therapy (by X-ray and radium) remain the standard forms for treating most types of cancer. New refinements of these techniques have saved the lives of many victims of cancers that hitherto had killed all their victims.

Chloromycetin and aureomycin shared top honors among newly developed drugs. Both are antibiotics, like penicillin and streptomycin. Both are cousins of streptomycin, being extracted from the same species of soil mould, streptomyces. Chloromycetin was first extracted from the soil of Caracas, in Venezuela. It was sent to Dr. Paul R. Burckholder of Yale University, who isolated the antibiotic. The drug was developed chemically in the Detroit laboratories of the Parke, Davis and Company drug firm. It was first used clinically on disease-ridden humans by U. S. Army medical research teams in widely separated parts of the globe including the Malay Peninsula, the Philippines, Mexico and the United States. Like that other infant drug, aureomycin, chloromycetin has proved effective in preliminary trials on diseases caused by rickettsia and viruses-microorganisms, smaller than bacteria, which have resisted treatment by the sulfa drugs, penicillin and streptomycin.

Scrub typhus, a disease that used to kill three out of every five victims, has apparently been conquered by chloromycetin. The new drug brought rapid recovery to the first 80 scrub-typhus victims on whom it was used. It is the first specific drug used effectively against typhoid fever. The drug has just gone into large-scale commercial production. It is taken by mouth, reduces high fevers with dramatic speed and apparently produces no toxic effects.

A number of serious and stubborn diseases began toppling before the on-slaught of the second big antibiotic find of 1948, aureomycin. Twenty-four patients suffering from acute brucellosis, or undulant fever, were treated successfully with the drug in Mexico by a team of Mexican and American medical researches. The treated patients must be observed closely for some time before the permanent effect of the new drug on this widespread disease can be evaluated. In aureomycin, doctors appear to have found their most effective chemical weapon against Rocky Mountain spotted fever, a serious rickettsial disease transmitted to man by a tick. Reporting in the *Journal of the American Medical Association* at the end of the year, six doctors

Geigy



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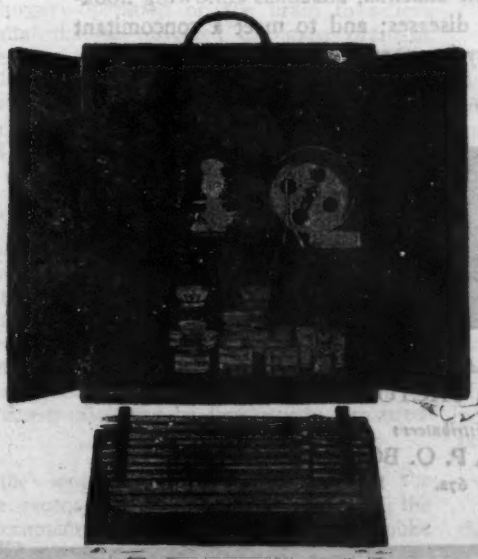

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connected with research centers in Baltimore and Washington, D.C., revealed that aureomycin treatment had proved very effective in each of 13 Rocky Mountain spotted fever cases. Whether aureomycin is superior to chloromycetin in treating this disease remains to be shown by further experimentation with both drugs.

Dr. Perrin Long of Johns Hopkins University reported successful treatment of virus pneumonia with aureomycin. A team of Columbia University and Harlem Hospital investigators in New York City revealed that aureomycin can cure lymphogranuloma, a virus-caused venereal ailment which has been one of mankind's most stubborn diseases. Two Columbia medical scientists found the drug very effective against several staphylococcal eye infections, curing 158 out of the first 200 cases treated. Many of these ocular diseases have been resistant to penicillin and streptomycin.

A new extremely important use was found for an older antibiotic, penicillin, during the past year. Doctors for some time have been using a single-injection treatment to cure gonorrhoea. Now it may be possible to prevent gonorrhoea by swallowing a single tablet of penicillin. A group of U. S. Public Health Service scientists performed the following experiment on a large number of military personnel: Members of certain military units were provided with penicillin tablets and told to swallow one within a few hours after each sexual exposure. Other units, used as control groups, went without the penicillin. The gonorrhoea rate among the latter was 508 per 1,000 men per year. The rate among those who swallowed the tablet shortly after exposure was only 105 per 1,000.

Psychosurgery-operation on the brain to relieve or cure mental disease provided the most dramatic news of the year in psychiatry. A group of New York and New Jersey medical researchers announced the development of a new brain operation, topectomy, which brought about the apparent recovery of 20 out of 24 schizophrenic patients who had spent an average of three to four years in a State mental hospital and whose cases had been considered virtually hopeless. Topectomy is a modification of the lobotomy operation. In the latter, the frontal lobes of the brain are pierced on both sides of the temple by an instrument resembling an ice pick. In topectomy, the frontal skull on both sides is opened and surface tissue is cut away in carefully mapped-out areas of the brain. Intensive follow-up study is being made of the patients who underwent the topectomy operation so that its permanent effect can be conclusively measured.

From the long-range viewpoint, probably the most notable recent psychiatry development was the Federal Government allocation of $6,000,000 to public and private agencies for the expansion of mental health clinics, for expanding the training of psychiatrists and for stimulating psychiatric research.

Millions of sufferers from hay fever and other allergic ailments were given the choice of a number of new drugs put on the market in 1948. Prominent among the new arrivals were trimeton, histadyl and antistine—all in the antihistamine family of drugs. These drugs could offer only relief—not cure—of hay fever and other allergies. On the whole they seem to possess no more potency than pyribenzamine, benadryl and neo-antergan, which have been available for several years. New evidence was produced during the year indicating that many allergy cases have a psychosomatic background; that is, they are influenced by mental and emotional factors.

Preventive dentistry has taken its biggest forward stride with the mass application of sodium fluoride to children's teeth. Seven years of carefully controlled experimentation have demonstrated that a two per cent solution of fluoride, applied four times to children's teeth, reduces dental cavities by nearly half. In many communities fluoride was added to drinking water as a measure of mass-scale prevention of tooth decay in children. No evidence has yet been produced that fluoride checks dental caries in adults. The past Congress appropriated $1,000,000 for Federal aid for the mass use of fluoride to combat tooth decay.

A mass preventive attack on tuberculosis was likewise launched with a Federal-aided program of BCG vaccination for 100,000 American children living in families, neighbourhoods and towns where the incidence of tuberculosis is especially high. Repeated mass-scale experiments have demonstrated that, although it is no certain preventive, BCG does drastically reduce the tuberculosis threat when used judiciously.

A major event was the National Health Assembly held in May 1948 in Washington under auspices of the Federal Security Agency. Eight hundred delegates, representing organized medicine, organized labour, public health, farm and consumer groups in the United States, met to draw up programs for solving the major problems of health and medicine. Mainly as a result of this Assembly, Federal Security Administrator Oscar R. Ewing submitted a report on the nation's health to President Truman, outlining a 10-year plan for filling unmet health and medical needs. Among other recommendations, Mr. Ewing proposed the creation of a compulsory national health insurance system which would provide comprehensive medical services for nearly the whole American population. This feature of his report promises to become a major issue in the present Congress.

Regardless of existing gaps and defects, however, the Metropolitan Life Insurance Company was able

to report that 1948 had been the healthiest year in American history, with a record low death rate somewhat under 10 per 1,000 population.—*U. S. Information Service.*

AUSTRALIAN STATE HAS TEN-YEAR PLAN FOR HEALTH

Geoff Taylor writes:—Concrete is flowing, bricks are being laid and steel fabricated as one of Australia's most ambitious hospital construction projects emerges from plans first conceived in the State of Victoria, in 1938-39.

Shelved during the war, the project has now been approved by the Hospitals and Charities Commission of Victoria.

The ten-year, three-stage plan involves expenditure of from Rs. 15,00,00,000 to Rs. 17,00,00,000 of which Rs. 4,00,00,000 is now being spent on the first stage.

Frequent surveys of the hospital resources and requirements of the State will ensure that, when completed, the project will not be out of date.

Architects responsible for the planning have kept abreast of post-war trends in world achitecture.

The full cost of public-ward construction will be met jointly by the State and Federal Governments, while the cost of construction to benefit non-public patients will be shared by the Victorian Government and local communities.

Where there is a need for a greater range of free services, the cost to the State Government will be greater.

De-centralisation—A feature of the big project is that Victoria has abandoned the once-accepted policy of concentrating hospitals in city areas. The plan envisages a chain of 300-bed hospitals throughout the metropolitan area of Melbourne. Six suburbs have already been chosen as dispersal points for the city's future hospital resources. Laid down in the original plans of 1938-39, this principle of de-centralisation was confirmed by subsequent war-time experience.

The planned development, by State authorities, of up-to-date hospital resources in suburban areas is a brekway from traditional planning.

Each Melbourne's new suburban hospitals will be fitted with complete radiological, pathological and physiotherapy, units together with birth-room suites, nursery units and casualty and out-patient accommodation. Indicative of the need for bolder plannig is that the highly-industrialised and thickly populated municipality of Footscray—Melbourne's equivalent of Birmingham or the U.S.A.'s Pittsburgh and now to be the site for one of the new suburban hospitals—was previously only served by 16 private-hospital beds. Footscray's will be the first completely air-conditioned hospital to be built in Victoria.

Outside the Melbourne metropolitan area, the State has been divided into regions, each to be served by a base hospital, where specialised treatment will be possible. Ten of these are to be developed in provincial cities or at other appropriate centres of Victoria's rural population. Subsidiary to each base hospital will be district and cottage hospitals. Medical services at these will be largely of the type associated with general medical practice.

Diagnosis Bureaux—The Victorian Government is now considering the establishment of medical diagnosis bureaux. These would be associated with the development of free dispensaries, home relief agencies, schools for the post-graduate education of nurses, provision of special accommodation for emergency cases and the setting up of a medical centre close to the University of Melbourne.

Marked decreases in diphtheria and tuberculosis have been recorded in Victoria over the past four years. The general trend of incidence of diphtheria in Victoria for the past decade has been downwards, the incidence during the past three years being the lowest ever recorded.

Active immunisation of Victorian school children against diphtheria began in 1923 in Bendigo, 100 miles north-east of Melbourne, when 40 per cent of the town's youngsters were tested and immunised. During 1947-48 approximately 34,000 Victorian school children were immunised—9,000 more than for the eight years up to 1934.

It is now hoped that equally satisfactory results will be shown with the State's anti-tuberculosis campaign, plans for which were recently presented to the Victorian Government and which are now being examined in detail.

Under Federal legislation passed last year, all States will now be reimbursed for capital and maintenance expenditure beyond that spent on anti-tuberculosis work in the base year ended June 30, 1948. Victorian figures for the period were approximately Rs. 30,00,000. Victoria has now outlined a comprehensive campaign to embrace general administration, expansion of case finding activities, improvement of clinical-treatment resources and development of libraries, statistics and publicity.

Extensive Testing—Victoria's campaign was recently extended to the detection of those who had been exposed to tuberculosis and the following-up of cases to locate sources of infection.

BCG (Bacille Calmette-Guerin), a vaccine not available in Australia, was recently imported from

Montreal, Canada, for the vaccination of medical and nursing staffs of Victorian sanatoria and hospitals. As BCG has to be used within ten days of leaving the laboratory and the trans-Pacific air-freighting took at least six days, careful organisation was necessary to ensure effective use of the previous vaccine on its arrival. Approximately 2,000 medical and nursing staff members were vaccinated in 12 Melbourne metropolitan hospitals, 13 provincial hospitals and three sanatoria. Large-scale vaccination will be possible when local supplies of the vaccine are available.

During 1947-48 over 23,000 Victorian school children were tuberculin-tested by the Vollmer patch-test method. The patches were prepared at a State sanatorium by means of patients under the direction of a skilled laboratory technician, himself a patient. In all, there were 1,028 positive reactions to the tests. Such a low percentage—4·3—made it easier to trace the sources of the children's contacts. Over 24,000 chest x-ray examinations were also carried out at five fixed centres.

There will soon be more than 2,000 beds available for tuberculosis patients in Victoria.

The State's medical authorities, however, will not be satisfied until each one of those beds is empty and superfluous.

Infant Welfare—On maternal and child welfare services, Victoria is planning further development. There are 366 infant welfare centres in the State, as well as ten ante-natal supervision centres and 14 circuit centres. One infant welfare centre has been established as part of a community centre scheme embracing infant welfare centre, kindergarten, playgrounds and mothers' club.

During 1947-48, 989,490 visits were made to centres by 7,232 expectant mothers who had 20,366 consultations. Nurses paid over 87,000 visits to homes.

Rural extension of infant welfare work has been accomplished by the formation of new country centres, provision of motor transport for country centre nurses, mobile services, and a correspondence scheme.

A travelling motor centre staffed by two nurses travels between a number of country towns. Travelling one-burse "circuit" services have been established to cover isolated areas of the State.

There are four infant welfare training schools for nurses in Melbourne.

In accordance with plans laid down in 1944 for the setting-up of a pre-school section, the State Department of Health is now planning a programme of health and education for all of Victoria's children in the age group "nought-to-six".—*Australian News Letter.*

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors.—EDITOR.

MALARIA AND PROBLEM OF ITS CONTROL IN WEST BENGAL

Sir,—It is needless to point out the wide prevalence of malaria in West Bengal.

In West Bengal annual deaths from malaria alone comes to 1,11,000 out of a total population of 2·11 crores. Considering 100 sickness for every one death, total number of persons suffering from malaria alone comes to 1,110,000 per year.

Economic losses due to deaths, treatment cost and loss of working days of workers, funeral cost, loss of efficiency in work when worked out comes to a fabulous sum.

Sinton worked out these costs for whole India which comes to Rs. 10,850 lakhs annually. (Rs. 108·5 crores annually).

Bengal as a whole was not so malarious a few decades ago. How it spread and assumed its present menacing form is known to all malariologists. There is no need further to stress upon the evil influence of this malady.

Wide differences exist between the malarial conditions in different localities within the same country. In West Bengal there are 4 zones divided from the point of view of malaria-carrying species of mosquitoes.

1. Sub-Himalayan West Bengal—*A minimus* breeds in slowly moving stream water, irrigation channels etc.

2. Coastal area—*A. Sundaicus* breeds in brackish water with algae, tanks, lagoons; may breed also in fresh water in coastal *beels*.

3. Cities like Calcutta—*A. Stephensi* breeds in wells, cisterns, fountains etc.

4. Rural Deltatic Bengal—*A. Philippinensis* breeds in tanks, pools, *dobas*, burrow-pits with vegetation.

Apart from these there are some species of local importance only.

It is interesting to note that bionomics of these species are different and a certain set of factors must be present for breeding for each of these species. Malariologists and entomologists have worked it out almost in detail except in case of *A. phillipinensis*. *A. phillipinensis* is by far the major species having a wider sphere of influence in consideration to other species of malaria-carrying mosquitoes.

Existence of malaria in a community depends on existence of (1) human reservoir of infection, (2) a vector mosquito (3) a susceptible individual and (4) favourable temperature and humidity.

The guiding principle on which the anti-malarial measures are based is to strike at the above chain at any vulnerable point.

Among the causes which spread malaria temperature and humidity are beyond our control. Thus

our anti-malarial efforts will be directed to tackle the remaining three factors in the chain of malaria infection.

Leaving human reservoir of infection and susceptible individual aside, I shall discuss here about the third factor, *i.e.* extermination of the species of the anopheles which transmit malaria. It is not possible to exterminate all mosquitoes. What we can achieve is that we can reduce the density of mosquitoes to a figure which is ineffective for transmission of malaria. It is also not necessary to attack all species; only vector species should be assaulted (species sanitation). It has been stated earlier that very wide differences do exist between malarial conditions in different countries. In formulating control measures, sweeping generalization without proper study of malariogenic factors is likely to be followed by disastrous consequences.

A classical instance of this occurred in Malaya where clearing of jungles—which prevents breeding of shade loving. *A. umbrosus* provided good breeding conditions for *A. maculatus* which is much more a dangerous carrier in Malaya. It is also obvious from the fact that in West Bengal there are separate 4 zones and malaria-carrying anopheline mosquitoes breed under different conditions in each zone so it is not possible even in West Bengal to make one sweeping generalization for control of malaria.

In different countries where malaria is present there are striking variations in climatic conditions, agriculture and modes of living of the people. This fact remains to be one of the great stumbling block in the anti-malarial works.

To ascertain these factors malaria survey is essential.

Survey—Malaria survey will necessarily be made before taking permanent measure for controlling malaria. This includes determination of

(1) Intensity of malaria.
(2) Time of the year during which it is prevalent.
(3) Distribution of malaria.
(4) Identification of vector species (species of mosquito responsible for carrying and harbouring infections) and
(5) Bionomics of the vector species.

In 1845 Surgeon T. E. Dumpster investigated the cause of unhealth in the canal irrigated zone of Western Jamuna canal. He employed the method of finding out percentage of persons with enlarged spleen. It is interesting that Surgeon Dumpster's method remains till to-day the only method for us to determine the amount of malaria present in a locality. Dumpster's report was illustrated by a map showing canal irrigated villages with high spleen rates and non-irrigated villages in the same tract with low spleen rates. It leads as to that the conditions which are produced by canal irrigation was responsible for high incidence of fever (malaria). Surgeon Dumpster worked out for the whole area—irrigated by the Western Jamuna canal. It is an interesting and valuable piece of work in the history of malariology.

We are now thinking of taking up anti-malarial measures of permanent nature on a very scanty material available at present. It is worth while to take up malaria survey of the regions by experts. It may be that time will be necessary for completion of work which will be entrusted to the experts to ascertain and study different malariogenic factors. But money and time spent will be the foundation of our knowledge for formulating permanent measures.

The Malaria Institute of India, only of its kind in the East is situated at Delhi which carries fundamental research work on malaria. Both laboratory and field station is now at Delhi with South India Branch at Madras. It is desirable that a Field Station may also be established at Calcutta for East Indian regions to study its special malariogenic factors.

Short term solution—Meanwhile measures directed against (*a*) the parasites inside the human host—both reservoirs and susceptibles in the form of distribution of anti-malarial drugs, such as Paludrine, Cinchona products etc., and also (*b*) against the parasites inside the insect host—spraying of houses with D.D.T. with residual spraying may be undertaken to control malaria as an interim measure along with the operation of keeping tanks clear in rural Bengal. I am etc.,

BIRAJA GOBINDO DAS, M.B., D.P.H.,
Berhampore,
West Bengal.

REVIEWS

ALL-INDIA MEDICAL DIRECTORY & WHO'S WHO—with a foreword by Dr. Jivraj Mehta, 1949. Demy 8vo., pp. ix+393. Published by the Health Book Stall, 7/8, Krishnan Koil Street, G.T., Madras. Price Rs. 20/- only.

This volume is perhaps the first of its kind in India and as is common to all pioneers, had to face a very large amount of difficulties and obstacles. It can easily be understood that the collection of the various types of data covering a wide range of fields must have called for a very good amount of painstaking energy on the part of the publishers. Considering all this, it must be said that this book has been commendably produced. The division of the subject matter into several sections from A to H has made the collated subject matter easier to follow, and refer to, in case of necessity. The general portion in section 'A' contains many points of interest, collated into a continuous chain by piecing together numerous informations from various sources. Sections B and E contain useful informations on various subjects suitably grouped. Section D has not come up to the desired standard, but this is only to be expected, considering

the great difficulty one has to experience in bringing together sufficient informative data covering detailed information about tens of thousands of medical men throughout the length and breadth of India. It is to be hoped that this section would appear in more detail in the next edition.

The printing, paper and get-up is good and the book removes a long felt want and hence is expected to be very much in demand.

R. SINHA.

THE 1948 YEAR BOOK OF OBSTETRICS AND GYNECOLOGY. Edited by J. P. Greenhill. pp. 603. Published by Year Book Publishers, Inc., 304, S. Dearborn Street, Chicago 4, U.S.A. Price $4·50.

This volume has well-maintained the tradition of its predecessors being ably edited for the last few editions by the present editor since the demise of late Dr. J. B. De Lee. The assorted 20 questions on the cover jacket give a welcome fillip to the enthusiasm of the inquisitive reader who finds a nice presentation of the entire subject all throughout the book. The abstracts have been well-chosen and comprehensive, further enlivened by personal remarks of the editor backed by his wide experience. References are found abounding in the book and are mostly, but not entirely, of American journals. A wider use and presentation of references from other countries would certainly make the future volumes still more useful.

These volumes have earned a well-deserved place in the library of every doctor who wants to be well-posted with the advancements made year after year; and, when the year ends, a fresh volume is eagerly awaited. This present volume has kept this up and is well got up, apparently bereft of wartime limitations.

R. SINHA.

ECTOPIC PREGNANCY—By K. M. Masani. Pp. vii+165. Published by the Popular Book Depot, Lamington Road, Bombay 7. Price Rs. 12/-.

This book written by an obstetrician of eminence and great experience of Bombay deals with a subject which is fascinating, often difficult to diagnose, and confusing on occasions. The subject matter has been dealt with elaborately being suitably divided into seven principal chapters. An additional eighth chapter contains the descriptions of many cases of ectopic pregnancy, picked judiciously from available literature and some of the author's own; these make this chapter very interesting and depicts the fascinating nature of the subject.

The subject matter has been very ably dealt with; there are plentiful references at the end of each chapter; printing is good with types that do not strain the eyes, paper is excellent and so is the get-up. A larger number of diagrams and photographic reproductions would make the book much more attractive.

R. SINHA.

MEDICINE AND HEALTH IN THE SOVIET UNION—By Henry E. Sigerist. Pp. xviii+364. Published by Jaico Publishing House, 28, Rampart Row, Bombay. Price Rs. 12/-.

The author of the book is reputed as a foremost medical historian. He has earned well-merited fame as the author of "Man and Medicine", "The Great Doctors", "Socialized Medicine in the Soviet Union", "Medicine and Human Welfare" and other books. He has made a personal study of the aspect of Medicine and Health in the Soviet Union and has undertaken extensive studies for this.

In this book Dr. Sigerist, in a vivid and graphic way, describes the back ground, concept, structure and practice of medicine including prevention and treatment of diseases, as well as of preservation and promotion of health—as he has seen and understood it to be prevalent in the Soviet Union of to-day. The book is well-written with understanding and knowledge based on extensive personal studies, and should be read and re-read by all those who are interested in Health Welfare and Health-planning for any community or country.

The paper, printing and get-up are very good.

R. SINHA.

HANDBOOK OF SURGERY—By Eric Mekie and Ian Mackenzie. Second Edition, 1949, Crown 8vo, pp. xvi+764. Published by E. & S. Livingstone Ltd. 16 & 17, Teviot Place, Edinburgh. Price 20 s. net.

This book, as the author of the first edition says, contains "the salient features of the subject which must be known ere the student presents himself for examination." In addition to the salient features, this handbook contains enormous number of facts which a student of to-day must know.

The only defect of this book is the want of sufficient number of illustrations. There are only 29 illustrations which seems to be too meagre for a text book of surgery.

However, there is much to be said in favour of this work. We can confidently recommend this book to every medical student.

K. DAS.

TEXT BOOK OF MEDICAL TREATMENT—Edited by D. M. Dunlop & others. 5th Edition, 1949. Royal med. 8vo, pp. xvi+999. Price 35 s. net.

The new edition of the too well-known book on treatment will be received with enthusiasm by the medical profession. The volume has maintained the high standard of previous editions. New chapters on antihistamine drugs, treatment of dehydration, care of the old have been included. The book is well arranged and contains contributions by many distinguished and experienced authors and above all has not been made unnecessarily voluminous. The table at the end of the book showing the proprietary equivalents of the official preparations will be helpful. This text-book on treatment can be recommended for the students and practitioners with confidence.

S. C. CHATTERJEE.

CRITICAL STUDIES IN NEUROLOGY—By F. M. R. Walsho, 1948. Royal 8vo, pp. xv+256. Published by E. & S. Livingstone 16 & 17 Teviot Place, Edinburgh. Price 15s. net.

Dr. Walshe has a style of his own which can hardly be imitated. His writings are sound, carry conviction, and his choice of expression is particularly to be admired.

In this little volume has been reprinted six of his papers on—cutaneous sensibility; giant cells of Bentz, motor cortex, pyramidal system; the mode of representation of movements in the motor cortex; role of pyramidal system in willed movements; the integration of medicine.

The student of neurology will find much food of stimulation of his thought in these writings and will also learn how best to corelate the observations of the workers in the different fields. To make a critical study of this volume on 'Critical Studies in Neurology' one would require a sound knowledge in anatomy and physiology of the nervous system.

S. C. CHATTERJEE.

A WAY TO NATURAL CHILD BIRTH—By Helen Heardman, 1948. Double Crown 16vo, pp. vii+124. Published by E. & S. Livingstone Ltd. 16 & 17 Teviot Place, Edinburgh. Price 7s. 6d.

This small book describes in details the exercises that a mother should be taught during antenatal period so that she can have a comparatively painless childbirth and can safely proceed on to successful breastfeeding. We recommend this book to all who are in charge of pregnant women.

N. G. MOJUMDAR.

TEXTBOOK OF THE RHEUMATIC DISEASES—Edited by W. S. C. Copeman. Compiled by 24 contributors. 1948, Royal med. 8vo, pp. viii+612. Published by E. & S. Ligingstone Ltd. 16 & 17, Teviot Place, Edinburgh. Price 50s. net. 1948.

This encyclopedic work is divided into 30 chapters and deals "Rheumatology" from all its aspects. The subject matter treated ranges from "historical" to "psychiatric aspect" and "social and industrial aspect." In the first chapter is discussed the vagaries of nomenclature and the Editor presents a clinical classification, which is practical and should useful. Two interesting chapters on "the Anatomy and Physiology of Pain" and "The Clinical approach to Pain" have been contributed by J. H. Kellgren.

This book is not a "Recent Advance" book, hence newer approach for treating rheumatoid arthritis is not mentioned.

To one who wants to gain an over-all picture of rheumatology this book forms a sure guide.

N. G. MOJUMDAR.

CURRENT MEDICAL LITERATURE

SYNDROME OF DIABETES MELLITUS AND ITS CAUSES

HIMSWORTH (*Lancet*, 1:473, 1949) writes: It is rarely possible to define a disease precisely but it is always possible to characterise a syndrome. In medicine definition entails elimination, for a syndrome is usually first glimpsed in a complex condition, associated with secondary syndromes, from which it has to be disentangled until its essential features are clear. The one essential of the syndrome of diabetes mellitus is an elevation of the blood-sugar level above those limits we regard as normal. On this depend glycosuria and, when its degree is sufficient, polyuria and thirst. But none of these dependent manifestations, nor any others in diabetic patients, are peculiar to diabetes mellitus. And the syndrome can arise in the absence of wasting, ketosis, or any evidence of failure to oxidise carbohydrate. Its one essential criterion is abnormal hyperglycaemia.

The causes of abnormal hyperglycaemia fall into two broad groups according to the site of the primary fault: those in which the primary fault is so situated as to impair the direct line of carbohydrate metabolism; and those in which initially there appears to be no such impairment but rather an incapacity to store ingested carbohydrate as fat or glycogen. In the first group are placed primary deficiency of insulin and inhibition of insulin consequent on overfunction of the anterior pituitary gland. In the same group would be placed any interference with the chain of intracellular carbohydrate oxidation—*e.g.*, hyperglycaemia of experimental thiamine deficiency. The second group would include the abnormal hyperglycaemia of those obese diabetics whose syndrome disappears on weight reduction and that which accompanies hepatectomy whether produced surgically or by tumours. It is not yet clear how several abnormal hyperglycaemias are to be classified. That associated with over function of the cortex of the suprarenal gland will probably fall into the group due to direct inhibition of insulin. The ultimate significance of the prediabetic state which exists in some pregnant women remains to be seen. The position of the hyperglycaemias due to infection and to various poisons can only be settled by further data. But, irrespective of the location of the primary fault, the abnormal hyperglycaemia common to all can lead to islet damage and secondary insulin deficiency, and so to the supervention, or aggravation, of an impairment to the direct metabolism of carbohydrate by the cells.

All the primary faults, save one, would cause insensitivity to insulin. The exception is deficiency of insulin. The results of the insulin-glucose test show that, whereas the sensitive response is the rule in the younger age-groups, the insensitive response is usually obtained in the middle-aged and elderly. The incidence of diabetes increases rapidly in middle age; the insulin-insensitive type of diabetic is therefore the most common in any representative diabetic population.

It thus appears that we should accustom ourselves to the idea that a primary deficiency of insulin is only one, and then not the commonest, cause of the diabetic syndrome. That is not to say, however, that in the other types of case insulin therapy is irrational. Insulin is our only means of breaking down any excessive inhibition of the process by which glucose enters the metabolic stream. Within the cell we are deal-

ing with a chain of reactions; and, provided the level of any particular enzyme is not reduced below a minimum, increasing the concentration of the incoming substrate may be expected to whip up the rate of turnover. But there are rare cases in which insulin fails. In them the primary fault is presumably of too great a degree to be overbourne by insulin. It is only by discovering the nature of such primary faults that we can hope to improve our therapy and, more important, to prevent the diabetic syndrome.

Low Blood Pressure in Diabetic Coma

Howarth and others (*Clin. Sci.*, **6**:247, 1948) write that it has long been recognized that in the later phases of diabetic coma the blood pressure may fall to low levels, and that this change is often irreversible, death following within a few hours. Efforts to raise the arterial pressure by drugs have been on the whole unsuccessful, while large intravenous infusions, given in the belief that the falling blood pressure was due to a diminished blood volume and a decreasing cardiac output, have frequently resulted in pulmonary œdema.

The low blood pressure in diabetic coma is due to a decreased total peripheral resistance, which is below 50 per cent of the normal value. The site of the vasodilatation has not been determined. The cool, pale skin suggests that skin blood flow is diminished. Attempts to combat the peripheral vasodilatation by constrictor drugs have so far failed. The vasoconstrictor drugs, pitressin, digitalis, and d-N-methyl amphetamine hydrochloride (Methedrine), fail to raise the low arterial pressure, or show only a small transient effect. The vessels may show a transient initial constriction, but then appear to become insensitive to further injection of the drugs.

Retinal and Vascular Damage in Diabetes

Croon and Scott (*Lancet*, **1**:555, 1949) write that a series of 60 patient who have had diabetes from fifteen to twenty-six years have been examined clinically and ophthalmologically and the following observations are made:

Vascular and retinal complications are not inevitable, even as late as twenty-six years after the onset of diabetes.

Clinical evidence of diabetic intercapillary glomerulosclerosis, as shown by the development of a nephrotic syndrome, was found in only 2 cases.

Diabetic retinopathy was observed in some cases without signs of hypertension or of generalised arteriosclerosis.

Arteriosclerotic changes in the retinal vessels were either absent or minimal in most of the cases with retinopathy, and 9 of the 18 patients with retinopathy had normal retinal arteries. It, therefore, seems that some factor other than arteriosclerotic change must be mainly responsible for the production of retinopathy in diabetes.

There did not appear to be any correlation between the vascular or retinal changes and the severity or control of the diabetic state.

Implantation of Insulin in Diabetes Mellitus

Vaŝgas (*Lancet*, **1**:598, 1949) writes that the application to human diabetics of the results of the corresponding experimeints on animals has been tested.

In the seven cases presented here it was shown that pellets of protamine-zinc-insulin-cholesterol, implanted subcutaneously, are absorbed as slowly as fat-soluble hormones.

The dose used in the implant varies from 20·0 to 92·7 units of unsulin per kg. of body-weight, except in one case in which 450 units was given.

It was calculated, in accordance with experimental data, that the daily absorption of the implant was about 1 per cent., which seems to be confirmed by the present cases. This assures a maximum duration of activity of the implant of about 100 days.

The authors consider that the method of implanting pellets of insulin used by them is extremely promising and worth further study with a larger number of patients.

Diabetic Coma

Lee and others (*Brit. M.J.*, **1**:565, 1949) from a clinical study of 28 consecutive cases of diabetic coma treated with and without the early administration of glucose observe:

Ten cases received glucose and 18 only saline in the first four or six hours of treatment. The two groups of cases were comparable.

There was a mortality of 40 per cent. in the glucose cases and of 11 per cent. in the saline cases.

The results of this study indicate that the administration of glucose to serious cases of diabetic coma in the early stages of treatment intefers with rapid rehydration and increases the mortality significantly.

Extreme Bradycardia

Doane and Krasnoff (*Am. Heart J.*, **36**:777, 1948) report on a patient with complete heart block whose ventricular rate during the last twelve hours of his life varied from 12·5 to 9·8 beats per minute.

The probable causative pathologic factor in this case was shown to be calcific deposits involving the A-V node and the right and left branches of the bundle of His.

It appeared that ephedrine was a useful drug in increasing the ventricular rate and in lessening the frequency of Stokes-Adams syndrome.

This is apparently the slowest sustained heart rate recorded electro-cardiographically in man.

Immediate Sequelae of Myocardial Infarction

Selzer (*J.Am.M.Sc.*, **216**:177, 1948) reports on the observations of a series of 130 unselected cases of recent myocardial infarction found at autopsy. In 35 patients myocardial infarction was a terminal event in otherwise seriously ill patients and was regarded as of little clinical interest. The remaining 95 patients were apparently well prior to the onset of the myocardial infarction and were subjected to detailed analysis as "primary" myocardial infarction.

The immediate cause of death in this group of 95 cases was: progressive circulatory failure, with or without shock, in 28 cases; sudden death due to arrhythmia in 24 cases; embolic phenomena in 15, cardiac rupture in 8 cases; secondary thrombosis in 5 cases. In the remaining 15 cases death was due to incidental complications not related to myocardial infarction.

No significant correlation was found between the age of the patients, the degree of the coronary arteriosclerosis, the size of the infarction and the presence of myocardial scars and of cardiac hypertrophy on one hand, and the course, the duration of illness, and the frequency of complications on the other hand. This is interpreted as indicating the adequacy of

circulatory adjustment even in cases with extensive damage to the heart.

The prognosis of acute myocardial infarction is unpredictable because the importance of cardiac insufficiency—the direct consequence of the damage to the myocardium—is outweighed by secondary complications. The immediate mortality is twice, or perhaps 3 times greater than the estimated number of cases with irreparable cardiac damage. The important sequelae of myocardial infarction, which can be considered potentially preventable cause of death, are: serious arrhythmia, thrombo-embolic phenomena and shock.

Veratrum Viride in the Treatment of Essential Hypertension

Freis and Stanton (*Am. Heart J.*, **36**:723, 1948) write that veratrum viride in the form of the whole powdered mixture of alkaloids was administered orally to a series of forty patients with essential hypertension for periods up to thirteen months.

When the drug was administered by the oral route, the hypotensive effect began to appear at the end of one or two hours, reached a maximum in four or six hours, and largely disappeared by the end of fourteen hours. In order to obtain maximum therapeutic benefit and to avoid toxic reactions resulting from cumulative overdosage, veratrum was administered at dosage intervals of twelve hours. In addition, in order to provide greater therapeutic safety, this dosage interval was further subdivided so that no more than 10 Craw units were ingested per hour. The great variability of response to a given dose in different individuals required the gradual increase of dosage in each patient in order to avoid toxic side effects.

Veratrum was found to be a useful therapeutic agent in the treatment of patients with (*a*) "hypertensive crisis", (*b*) severe, long-standing hypertensive disease which proved resistant to other forms of treatment, and (*c*) hypertension complicated by cardiac failure.

Prolonged therapy in some cases resulted in a diminution in cardiac size and reversal of electrocardiographic changes toward normal.

There were no deaths and no toxic reactions resulting in more than transient disability attributable to the drug. However, the development of side effects and of changing sensitivity to a given dose were sufficiently frequent to limit its usefulness in the treatment of patients with mild or moderate degrees of hypertension.

Veratrum viride appeared to have therapeutic value as an adjunct to dietotherapy and to the routine treatment of hypertensive heart diseases.

Neurological Manifestations of Digitalis Toxicity

Battelmann and Gutner (*Am. Heart J.*, **36**:583, 1948) write that the neurological manifestations of digitalis intoxication are more common than has been appreciated previously. Approximately 41 per cent of toxic symptoms involved some portion of the central or peripheral nervous systems. Gastrointestinal complaints are only slightly more common but it is our belief that the incidence of their occurrence and the importance of their recognition as indications of digitalis toxicity have been overemphasized.

The ten patients presented exhibited unusual neurological complaints. A careful survey of the literature failed to disclose any previous recording of such symptoms. It is of interest to note that these complaints occurred in approximately 9 per cent of patients manifesting digitalis toxicity.

The neuralgic type of pain usually involved the lower one-third of the face simulating the syndrome of trigeminal neuralgia. The pain was characterized usually as a dull aching in the teeth or as a sharp and stabbing pain throughout the mandible or maxilla, or both. Other areas involved with typically neuralgic "shooting pains" were the upper extremity, lower lumbar area with posterior thigh radiation, and calf muscles. Parasthesias, such as tingling in the fingers and burning sensations in the feet, were also observed.

The type of digitalis preparation administered apparently played no role in the occurrence of these neuralgic complaints.

Apical Systolic Murmur

Master (*Arch. Int. Med.* **81**:518, 1948, Ref. *Am. Heart J.*, **38**:843, 1949) believes and quotes numerous extensive personal and insurance statistics to support his belief, that loud apical systolic murmurs, even in the absence of cardiac enlargement, heart failure, diastolic murmurs, or abnormal electrocardiograms, are a sign of organic heart disease. The overall mortality for persons under 40 years of age with apical systolic murmurs is 3·25 times higher than the normally expected mortality rate, 4·5 times higher when there has been a history of rheumatic fever. The mortality rate is 50 per cent higher among manual workers with apical systolic murmur than among "white collar workers."

It should be evident then that the proper evaluation of the murmur is of paramount importance in medical practice both in war and in peace. Use of the term "loud apical systolic murmur" includes all grades of intensity except the extremely faint and slight and will include the moderately loud, loud, extremely loud, and unusually loud murmur (classification of Freeman and Levine). Physical examination, history and laboratory examination are still of major importance in the final determination. A history of rheumatic fever in a patient with a loud murmur should be accepted as almost certain evidence that a defect in the mitral valve exists. The presence of a "musical, harsh, sea-gull, or constant murmur" strengthens the diagnosis. Patients should be examined repeatedly, in different positions and after exercise, since the murmurs of early valvular heart disease are transient.

Exercise will not produce "loud" apical murmurs in healthy persons. The teleroentgenogram and fluoroscope are of considerable import in diagnosis, as is the electrocardiogram. The occasional difficult differential diagnosis from the "effort syndrome" can usually be made on the basis of other well-recognized symptoms of neurocirculatory asthenia.

The author believes that loud systolic murmurs at the apex should, therefore, be considered organic, and the patient treated accordingly, receiving antibiotics during various manipulations and diseases which injure the mucous membranes and facilitate the entrance of bacteria into the blood stream which may lead to bacterial endocarditis. Establishment of the organic nature of a murmur does not afford a criterion of the heart's function.